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A PRACTICAL TREATISE

ON THE

DISEASES OF THE HEART AND GREAT VESSELS.

A

PRACTICAL TREATISE

ON THE

DISEASES OF THE HEART AND GREAT VESSELS,

INCLUDING THE

PRINCIPLES OF THEIR PHYSICAL DIAGNOSIS.

LANE L

BY

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CONSULTING PHYSICIAN TO THE HOSPITAL FOR CONSUMPTION.

"Rerum ipsarum cognitio vera e rebus ipsis . . . ."—Jul. Scaliger.

FOURTH EDITION REVISED, AND MUCH ENLARGED.

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P. C. A. LOUIS

In Memoriam.

ADVERTISEMENT TO THE FOURTH EDITION.

The present edition has been carefully revised; much new matter has been added, and the entire work in a measure remodelled.

Some facts and discussions, more or less novel, will be found in the description of the principles of physical diagnosis, as applied to cardiac and vascular diseases. But the chief additions have been made throughout the clinical portions of the book; and some affections, of which little or no account had been given in the previous editions, are now treated of in detail. Functional disorders of the heart, the frequency of which is almost rivalled by the misery they inflict, have been reconsidered; more especially an attempt has been made to render their essential nature clearer, and, consequently, their treatment more successful, by an analysis of their dynamic elements.

While we are, on the one hand, justified in affirming that the medical and surgical means of successfully controlling the progress of structural diseases of the heart and great vessels have materially improved within the last few years, we can, on the other, lay claim to quite as positive advancement in their hygienic management. And hence it comes, that the hopeless motto of Corvisart's original and model work on cardiac diseases,—

"Hæret lateri *lethalis* arundo,"

infinitely well chosen in his time, has, at the present day, lost much of its telling appropriateness.

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A PRACTICAL TREATISE

ON THE

DISEASES OF THE HEART AND GREAT VESSELS.

. . . "Who knows but that one may discover the works performed in the several offices and shops of a man's body, by the sounds they make, and thereby discover what instrument or engine is out of order?"—R. Hook, 1705.

INTRODUCTION.

I.—CLINICAL TOPOGRAPHY OF THE HEART AND GREAT VESSELS.

1. The heart, seated in the lower part of the anterior mediastinum, is held *in situ* directly by the great vessels, arterial and venous, and, indirectly, through the pericardium by the diaphragm. These attachments, fixing the base only of the organ, permit free play to its general mass, which in point of fact hangs loosely in the pericardial sac. This looseness of attachment and this freedom of movement, essential to the physiological well-being of the central agent of the circulation, entail a specific inconvenience in the extreme facility with which the organ, in this respect almost rivalling the uterus, undergoes various displacements.

2. Lying obliquely (with its long axis directed forwards, downwards and from right to left, at a slightly varying angle with the mesial plane), the base of the organ corresponds anteriorly, to both third costal cartilages, and the apex to the sixth left costal cartilage; while, posteriorly, the upper edge of that base lies opposite the fourth, the lower opposite the seventh,

or even the eighth dorsal vertebra, separated from the spine by the aorta and œsophagus. A line, carried horizontally backwards from the apex, falls in the majority of adult males on a spot seated less than two inches to the left of the body of the eighth dorsal vertebra.

3. The postero-inferior surface of the organ, somewhat flattened, rests upon the central tendon of the diaphragm; the supero-anterior lies in apposition partly with the left and right lung, partly, between these organs, and especially at its lower part, with the walls of the chest.

4. Of the two main borders of the heart, the right, notably the thinner, lies lower, nearer the anterior chest-wall, and is habitually more horizontally placed and straighter than the left.

5. The heart occupies the entire of the lower sternal, with certain portions of the left and right mammary regions, and inclines to encroach, more or less according to the amount of repletion of its cavities with blood, on the lower aspect of the infra-clavicular regions—an encroachment practically more frequent and significant on the right than the left side.

6. The relationship of the different compartments and orifices of the heart to the surface is a matter of which the clinical student cannot exaggerate the importance; upon correct notions concerning this relationship essentially hinges the diagnosis not only of certain diseases of the heart itself but of neighbouring structures. The topographical statement I am about to submit for his guidance embodies the results of observations continued for a number of years, and made by driving long needles into the bodies of males, at various fixed points perpendicularly to the surface, before the abdomen or thorax had been opened. The subjects, thus treated, had been cut off by all varieties of disease indiscriminately: collation of the observations made upon the whole series at once showed, as might have been anticipated, that the cases bore division into four classes—(*a*) those in which the position of the organ was perfectly normal; (*b*) those in which intrinsic disease had perverted the relationships of its several parts; (*c*) those in which extrinsic disease had affected its topography; and (*d*) those in which intrinsic and extrinsic disease had both severally produced their effects. In the present place the normal state will alone be considered; and to facilitate the process of fixing the topography of the different spots, the subjoined rude diagram is offered.

The outlines and spots indicated only apply with precision to the male body examined in the recumbent posture, and free from any notable amount of physiological peculiarity either in the form of the chest, the volume of the lungs, or the bulk of the solid, and fulness of the hollow, abdominal organs. Nor must it be forgotten that the average result only is here presented,—on either side of which slight oscillations, in all conceivable directions, are compatible with perfect functional health of the thoracic organs.

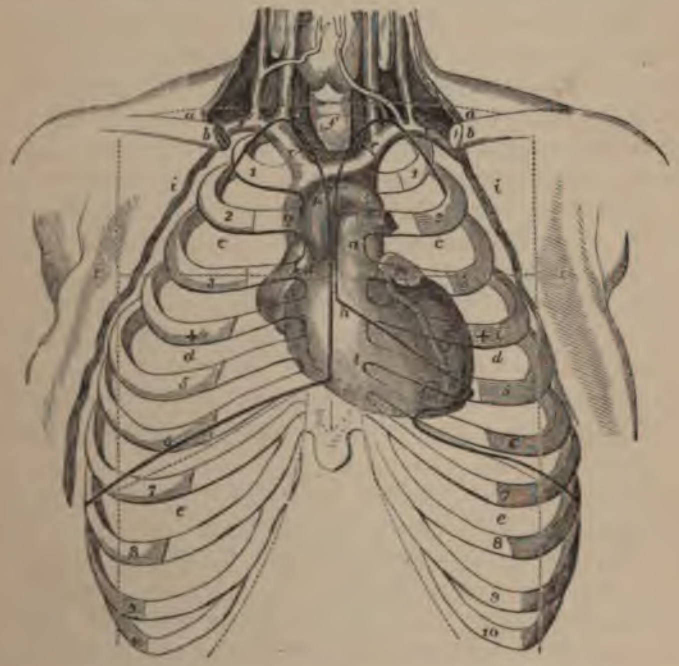

Diagram exhibiting the relationship of the heart and great vessels to the lungs (in moderate inspiration) and to the regions of the chest. 1 to 10 inclusive, ribs; *a*, supra-clavicular region; *b*, clavicular; *c*, infra-clavicular; *d*, mammary; *e*, infra-mammary; *f*, supra-sternal; *g*, upper sternal; *h*, lower sternal; *i*, integuments turned back; † †, nipples; *k*, right auricle; *l*, right ventricle; *m*, left auricle, appendix almost solely seen; *n*, left ventricle; *o*, pulmonary artery; *p*, arch of aorta; *q*, vena cava superior; *r r*, innominate veins; *s*, innominate artery; *t t*, subclavian veins. *The dotted lines indicate the outlines of the thoracic regions; the dark lines the edges of the lungs. The heart and vessels are supposed to be full.*

The site of the various spots given in the text was ascertained by driving needles vertically through the body at different places on the surface of the chest. In order as far as possible to prevent the influence of pulmonary collapse in altering the position of parts, the lungs were gently and moderately insufflated and the trachea firmly tied, before the thorax was opened.

7. (A.) *Relationships of the heart's compartments and valves and of the great vessels to the anterior surface of the chest.*—The *right auricle* reaches (more or less extensively, according to the amount of blood stagnating in or moving through the right side of the heart,) into the right mammary region,—mainly on the

level of the third cartilage and fourth interspace, slightly on that of the second space. In cases of extreme distension this auricle, reaching the fifth right cartilage below, may stretch upwards almost to the inferior edge of the second cartilage.

8. The *right ventricle* corresponds mainly to the lower sternal, and the left mammary regions. Its inferior, and nearly horizontal, border stretches from the fifth right cartilage to the point at which the apex beats in the fifth left space midway between the left edge of the sternum and a line let fall vertically from the left nipple, coasting, as it passes, the upper part of the sixth left cartilage. The vertical span of this ventricle at the left edge of the sternum reaches from the third to the sixth cartilage. The base of the ventricle, running slantingly from above downwards and from left to right, encroaches slightly (more inferiorly than superiorly) on the right mammary region, between the third interspace and fifth cartilage.

9. The *left auricle* lies deeply behind the root of the pulmonary artery,—little but the appendix (Diagram, *m*), being visible anteriorly, as the organs lie *in situ*. The middle point of this auricle, from above downwards, corresponds to the cartilage of the third rib; the auricle encroaches to a variable extent on the second and third interspaces.

10. The *left ventricle* reaching vertically from the third to the upper edge of the sixth left cartilage, occupies a portion of the left mammary region. A comparatively narrow strip only of this ventricle is visible anteriorly (Diagram *n*).

11. The *heart* then, *as a whole*, extends vertically, from the second space to the sixth cartilage; and transversely, from a little within the left nipple to a finger's breadth or more to the right of the sternum: these are the limits of the *deep cardiac region*. The entire of the left ventricle, the greater part, by far, of the left auricle, and a large portion of the right ventricle, towards the apex, lie to the left of the sternum; and on the level of the fourth cartilage, the widths of heart substance, lying on either side of the left border of the sternum, are very closely the same.

A line, carried directly backwards from the apex of the upper angle of the fourth left interspace, will fall as nearly as possible on the centre of the heart's surface.

12. The chief relationships of the lungs to the heart, applicable in clinical study, are as follow. The entire of the right auricle, and about one-third of the right ventricle, are covered by the right lung, which passes vertically down-

wards, coasting the middle line, to the lower edge of the heart: the entire of the left auricle, the upper left part of the right ventricle, and the entire of the left ventricle, except a variable, but small, portion towards the apex, are covered by the left lung. The portion of heart uncovered by lung, thus belonging almost exclusively to the right ventricle, is of rudely triangular shape. The upper angle of the triangle corresponds to the middle line on the level of the fourth cartilage, where the anterior edge of the left lung diverges from its fellow; while the base is formed by that portion of the lower edge of the heart lying between the middle line and the spot at which the apex beats; this is the *superficial cardiac region*. The vertical side of the triangle measures on an average, in the middle-sized adult, two inches,—the horizontal, two inches and a half,—the oblique, nearly three inches. (Vide Diagram.) But, although it be certain that the triangular outline is of sufficiently constant occurrence to be regarded as the typical condition, it must be admitted the form varies,—sometimes resembling a parallelogram, sometimes nearly square, sometimes wholly irregular.

13. *Valves.*—(*a*) The upper or free edge of the *pulmonary* valves lies horizontally, and in the mass of persons a shade above the upper edge of the third left cartilage, the body of the valve consequently a little lower than this,—the left edge of the sternum having closely the same width of the vessel on both sides. Luschka* places this valve somewhat higher; but, in point of fact, almost all the spots of importance are fixed by him at a slightly higher level of the chest, than would be justified by the average result of my observations.

(*b*) The *aortic* valves, lying horizontally a very little further inwards, and lower than the pulmonary, correspond to the union of the sternum and third left cartilage.

(*c*) The attached edge of the *tricuspid* valve, slantingly placed across the sternum from above downwards, and from left to right, inclines from the neighbourhood of the sternal edge of the third left interspace nearly to the sternal end of the fourth right interspace or fifth right cartilage.

(*d*) The attached edge of the *mitral* valve lies almost horizontally about one-quarter of an inch lower than the attached bases of the aortic valves, very slightly further inwards than these, and deeper within the chest than the tricuspid valve. The attached border lies on the level of the union of the third cartilage with the sternum, nearer, as a rule, the upper than the lower border of this: I have, however, found the body of the valve as low as the mid-height of the third interspace.

---

* Die Brustorgane des Menschen in ihrer Lage. Fol. Tübingen, 1857 (p. 12). This is a work of such carefully and conscientiously elaborated detail, that, when I find my own observations differing from those of its author, I have the most serious misgivings that truth is not on my side. Can it be possible, the slight discordances of statement, referred to in the text, may find their explanation in the difference of form, as an average result, in the German and English thorax? (Vide Diseases of Lungs, 4th Ed., p. 337.)

14. The transverse distance between the nearest points of the right pair of valves, tricuspid and pulmonary, averages half an inch; the left sets, aortic and mitral, fall within the same vertical plane.

15. A superficial area of half an inch square will include a portion of all the four sets of valves *in situ:* an area of about one quarter of an inch a portion of all except the tricuspid. Hence the isolation and distinction of the sounds and murmurs produced at the several orifices, if possible at all, must be effected on some other principle than that of direct conduction to the nearest spot of the chest-surface,—that spot is in point of fact practically the same for all four.

16. *Aorta.*—The aorta, rising from the sternal end of the third left cartilage, nearly opposite the union of the two sternal regions, ascends at once into the upper sternal region, at first under cover of the pulmonary artery; and, inclining to the right, reaches by its right border the inner and upper part of the second right costal cartilage, occasionally touching on the inner edge of the first interspace: thence crossing, almost horizontally, the upper sternal region, on the level of the first interspace, and in front of the trachea just above its bifurcation, it passes backwards and downwards to the left side of the body of the third dorsal vertebra. Sometimes the entire of the arch lies a little higher than this.

17. *Pulmonary Artery.*—The pulmonary artery ascends from the position of its valves, with slight inclination to the left side, as far as the second left cartilage or first interspace, its point of bifurcation; in this short course it passes somewhat backwards, and consequently lies further somewhat from the surface of the chest opposite the second, than opposite the third, cartilage. It occupies a portion of the upper sternal region, encroaching on the edge of the left infra-clavicular.

18. *Arteria Innominata.*—The arteria innominata rising on the level of the upper edge of the first interspace, or on that of the lower part of the first cartilage, behind the right half of the upper sternal region, passes upwards and to the right, bifurcating

to the right of the trachea, and behind, or a little above, and to the right of, the sterno-clavicular joint: in its course it lies partly in front of the right half of the trachea.

19. In as much as the sounds and murmurs produced in or transmitted along the arch of the aorta, the pulmonary and the innominate arteries are, as a rule, severally best isolated at the second right, the second left, and the first right, costal cartilages,—these cartilages may for clinical purposes be respectively called the aortic, pulmonary, and innominate. An important qualification to this general truth must, however, be added. In as much as the pulmonary artery at its root lies in front of, and nearer the surface than, the aorta, with which it stands in close union, it follows that at the actual base the vibrations of the aortic valves are not only conducted to the surface through the pulmonary artery, but having reached the walls of this vessel may be freely transmitted along them, and hence that either sound or murmur really produced at the mouth of the aorta may be better audible at the cartilage we have just named pulmonary, namely, the second left, than at the second right or aortic.

20. *Superior vena cava.*—The superior vena cava lies along the right edge of the ascending portion of the arch of the aorta, near the right border of the sternum, from the first interspace to the third cartilage; in the former spot occurs the union of the two innominate veins.

21. *Pericardium.*—The pericardial sac (representing a cone of which the base, lying inferiorly, is fixed to, and partly incorporated with, the upper surface of the central tendon and muscular portion of the left ala of the diaphragm, while the apex, placed superiorly, embraces the lower two inches of the great vessels) extends from the second to the seventh left costal cartilage. In rare instances, however, where the pericardium proves wholly free both from present disease and from anatomical evidences of past morbid distension, the cavity of the sac reaches the level of the first costal cartilage.*

A larger portion of the pericardial sac lies to the left than the right of the middle line;—the superficial areæ of the two portions standing to each other, very closely in the ratio of 3 : 2. Hence the extent of reflected pleura is greater on the left than the right side,—a fact wherefrom some diagnostic inferences indirectly flow.

The sac of the pericardium lies further from the anterior chest-wall superiorly than inferiorly,—a mode of relationship which lends its aid in rendering the detection of fluid less early, as a rule, at the upper than the lower part of the cavity.

---

* H. Hodson, U. C. H., Males, vol. ix. p. 20.

22. The precise position of the heart is influenced in a variety of ways independently of the intervention of actual disease.

(*a*) As has already been signified [6] the precise localization of the heart within the thorax varies, simply as a natural conformation, independently of disease either in, or beyond, the organ itself. This variation expressly affects height,—the organ lies a little lower or a little higher than natural.

(*b*) The action of the heart is accompanied, as vivisections prove, by alterations in form, size, axis, and position; in all three conditions—systole, diastole, and repose—the organ is specially characterised in regard of these four attributes. But to what extent are these changes, produced by the heart's own working, clinically appreciable? In the perfectly natural state not one of them can be substantiated except the visible and palpable alternate advance of the heart's apex to, and recedence from, the surface during systole and diastole. But in certain morbid states it may be ascertained, by the help of the eye and hand combined, that during the systole the organ twists spirally on its longitudinal axis from right to left, especially towards the apex, which at the same time comes forward [53, (2)]; while the converse movements attend the diastole. In certain cases, too, where obstruction to the intra-cardiac circulation is carried to a high point, alterations in size and even in shape may be established by careful percussion.

(*c*) Age exercises an influence in this direction. In early youth the organ lies higher than in the mass of persons advanced in life. Many conditions probably combine to lower its position as years roll on: the increasing weight and bulk of the heart itself, a normal effect of prolongation of life, drag it down; the pressure in aged people of emphysematous distension of the lungs pushes it down; and the aorta, athermatous or actually calcified in old age and hence impaired in elasticity, sustains the weight of the organ less effectively than in youth.

(*d*) The heart of the civilized adult female, of the present day, lies on the average slightly higher than that of the adult male. I am inclined to believe this difference the result of dress: the habit of tight lacing in the female will tend to lower or raise the heart according as the line of maximum pressure is low or high,—consequently, as the fashion of low waists prevails at the existing period, to raise it. The tight abdominal belt worn by some males exercises a similar, though less marked, influence.

(*e*) Alteration of posture of the body affects the site of the organ also. The heart falls downwards somewhat (if its substance be weighty, the fall may equal an inch) in the erect attitude, and comes more forward than in decumbency. Changing the posture in decumbency from the right to the left side will carry the heart an inch, or even more, to the right or left of the position it occupies when the individual lies on the back. Now this is a fact of considerable importance, as it shows that, especially when such change is only moderate in amount, lateral moveableness of the dull percussion-sound of the deep cardiac region cannot be accepted as conclusive of the existence of fluid in the pericardium. In the prone posture and in decumbency on the left side the heart comes more fully towards the chest-wall, than in the supine posture, or in decumbency on the right side,—facts which have their clinical application in the diagnosis of pericardial friction-sound.

(*f*) The mode of dissolution exercises an influence on the precise position of the heart *post mortem*. If the immediate mechanism of death has been asphyxial, the heart will lie lower than if it had been syncopal, and the amount of the difference will be in the direct ratio of the degree to which the asphyxial or syncopal phenomena have been severally carried. In comparing the position of the heart after death with that supposed to have been established during life, the mechanism of the final struggle must consequently be borne in mind.

(*g*) Locomotion of the heart attends each act of respiration. Inspiration, by carrying the diaphragm downwards, lowers the heart, sometimes by an entire interspace; and, by bringing a thick stratum of the left lung in front of the organ, removes it somewhat from the thoracic walls: the weakening effect thus produced on the heart's impulse and sounds is in healthy persons very perceptible. The position of the valves and the maximum points of the cardiac sounds are proportionably lowered; but it is to be remembered, that the depression of the diaphragm displaces the base more than the apex of the heart. This depression tends to lengthen momentarily the great vessels, especially the ascending portion of the arch of the aorta. The converse effects are produced by expiration. The amount of this locomotion is unequal in the two sexes. In as much as the calm breathing movement of the male is essentially abdominal and inferior costal (phrenic), while that of the female plays specially on the upper third of the chest, the heart changes its place more extensively with the respiratory act in the former than in the latter sex.

23. (B.) *Relationships of the heart's compartments and valves and of the great vessels to the posterior surface of the chest.*—On the whole, these are of much less clinical importance than the anterior group.

24. Of the heart itself the parts which come nearest the posterior chest-wall are the left auricle essentially, and the left ventricle to a small extent at its upper part. These, the portions of the heart lying furthest backwards, are separated from the surface by the pericardium, superiorly and transversely by the left bronchus, vertically by the œsophagus, the thoracic aorta and the vertebral column,—on the left side by the lung, while on the right (scarcely more than one quarter of the left auricle lies to the right of the mesial plane of the body) the lung is separated from the heart proper by the right pulmonary veins, vena cava inferior, and right main bronchi. As, anteriorly, scarcely any of the left auricle is visible on the surface, so, posteriorly, a very small portion only of the lower edge of the right auricle nears the surface.*

25. The descending portion of the arch of the aorta, continued into the thoracic aorta, passes on the left of the spinal column behind the left pulmonary artery, the left main bronchus, the left auricle, and the posterior base of the left ventricle. Murmurs produced in the course of the thoracic aorta are well audible in the left vertebral groove from the fourth to the eleventh ribs; those produced at the aortic valves and conducted in the walls of and within the vessel, are, as far as the back is concerned, best heard in the left vertebral groove from the third to the fifth spaces,—assuming that the conducting faculty of the lungs and other adjoining structures has undergone no deviation from the standard of health. Aortic or pulmonary murmurs are with difficulty transmitted in a direct line through the intervening tissues backwards to the posterior surface of the chest.

26. The mitral orifice corresponds most nearly to the level of the sixth rib and space in the left vertebral groove,—and here the murmurs produced at that orifice are best audible by direct conduction backwards.

27. The lines of attachment of the pleural laminæ to the sternum and chest-wall, where they turn off to form the mediastinum, vary very considerably. Thus both pleuræ may approxi-

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* Luschka, loc. cit. Taf. iv.

mate so closely, that the anterior mediastinal space becomes a mere fissure; or each lamina may actually fail to reach its own sternal margin, leaving a width of mediastinal space greater than that of the sternum itself—in fact, all imaginable varieties of arrangement, as has especially been proved by Bochdalek,* may be actually observed. One of the peculiar arrangements of greatest practical significance is that, where the mediastinal laminæ, widely apart at the chest-wall, converge in their course backwards towards the pericardium, so as to give the mediastinal space a triangular shape, with its base at the sternal surface.

II.—WEIGHT, SIZE, AND MEASUREMENTS OF THE HEART.

A.—IN HEALTH.

28. *Weight.*—In as much as the heart varies very sensibly in bulk with the stature and mass of the whole body, and also with the age of the individual, it follows that a general standard applicable with moderate precision to all persons, even of the same sex, is not attainable. For practical purposes, I have been in the habit of assuming (on the ground of results obtained during a number of years) that nine and a half ounces represent the average weight of the healthy male heart, eight and a half ounces that of the female organ, between the ages of thirty and sixty. A material deviation in either direction from these estimates would indicate failure or excess of nutrition, especially as they not only represent the mean, but the most common weights in a very large number of instances, where no evidence of cardiac disease, primary or secondary, had been clinically traceable.

Averages very notably higher than these have been given by other observers, especially by John Reid and Peacock: according to their tables, the most usual weights vary between ten to twelve ounces in the male, and from eight to ten in the female.

29. The attempts to fix a relationship between the weight of the heart and of the whole body have not led, as might be anticipated from the results just stated, to the establishment of any very fixed proportion,—the ratio varying from 1:158 to 1:176, according to different authors.

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* Vierteljahrschrift für die prácktische Heilkunde, 1er Band, 1860.

30. *Size.*—The measurements of the organ have been held to average vertically five inches, transversely three and a half inches, antero-posteriorly two and a half inches. The cavities should be freed as completely as possible from blood before the measurements are taken. Slight errors are easily made.

31. *Thickness of walls.*—The thickness of wall of the different parts of the heart has been most carefully established by Bizot,* and from his tables the following excerpt, having real practical significance, is arranged. It displays the mean thickness in French lines of the walls of the ventricles and septum, exclusive of the columnæ carnæ, between the ages of sixteen and eighty :

	MALES.			FEMALES.		
	Base.	Middle.	Apex.	Base.	Middle.	Apex.
Left ventricle .	$4\frac{68}{122}$	$5\frac{19}{122}$	$3\frac{23}{122}$	$4\frac{3}{8}$	$4\frac{4}{5}$	$3\frac{13}{30}$
Septum . .		$4\frac{5}{13}$			$4\frac{1}{36}$	
Right ventricle	$1\frac{113}{122}$	$1\frac{99}{244}$	$1\frac{3}{61}$	$1\frac{3}{8}$	$1\frac{7}{24}$	$0\frac{673}{720}$

Hence it appears, the middle part of the left and the base of the right ventricle are respectively the thickest parts. From other tables, it follows that, whereas the thickness of the left ventricle steadily increases with advancing years from puberty to extreme old age, that of the right ventricle remains almost stationary. The earlier the period of life, the less the difference between the thickness of the walls of the two ventricles.

The mean thickness of the wall of the right auricle has been estimated at about 1 line Fr. by Bouillaud,—that of the left 1½ line.

32. *Weight of heart.*—The discoveries, independently made by Bizot and Clendinning†, that the weight of the heart, the thickness of its walls, and the length and breadth of its mass, increase, as age advances, have their clinical application, especially in individuals of the male sex, in whom the change is much more sensible, both in amount and in steadiness, than in females. A weight over ten ounces in a male, aged five-and-twenty, would certainly render the existence of morbidly hypertrophous growth more than probable, whereas at the age of fifty a weight of ten ounces and a half might have coincided with a perfectly healthy state of the organ in all its local attributes and systemic influences. It must, however, be confessed that in the present state of knowledge there are intermediate degrees of over-weight, the healthy or mor-

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[*] Mém. Méd. Soc. d'Observation de Paris, t. i. p. 280. 1836.

[†] Med. Chir. Trans., 1838.

bid nature and tendencies of which can only be determined by reference to the past functional condition of the organ: in other words, it results positively from my observations that a given weight of heart, in a given sex, at a given quinquennial period of life, with a given stature, and a given weight of the whole body, will in one instance have belonged to an individual whose heart has never caused him the least trouble, in another to a patient who has suffered from many of the minor, perhaps some of the graver, evils of cardiac over-growth. Forced by experience to accept this conclusion, I attach little importance to slight excess (from half an ounce to an ounce and a half) above the alleged normal par of weight (*Vide* Appendix A).

33. *Capacity of Compartments.*—The absolute and relative capacities of the four compartments of the heart, during life, cannot be estimated with precision from examination of their areæ after death. The manner of dissolution must obviously affect to a serious degree the relative condition of the four cavities; and hence it is that the results of different observers vary so widely as to render them all untrustworthy. In regard of absolute capacity, nothing can be stated with even an approach to certainty.[*] The capacity of the two ventricles is probably equal,—that of the right auricle slightly, very slightly, greater than that of the left,—scarcely even in the proportion, taught by Cruveilhier, of 5 : 4.

34. *The four cardiac orifices.*—(*a*) In *shape* the arterial orifices have always been held to be round, the tricuspid and mitral openings elliptic. Herbert Davies,[†] however, shows that in infinite probability, to say the least, the latter pair of apertures are, *when in function*, circular, as well as the arterial.

(*b*) The measurement of the *width* or *circumference* of the four orifices of the heart, though by no means so easy as might on first thought be imagined, is not beset with the extreme difficulties attending that of the capacity of its cavities. The simplest and most correct plan consists in laying a wet piece of thin cord along the attached border of the auriculo-ventricular, and the free border of the arterial, valves: there is, thus, no necessity to stretch the parts, as the cord follows closely the inequalities of the surfaces.[‡] The mean measurements, as deduced by Bizot from observations on the bodies of persons aged from sixteen to eighty, are subjoined in French lines :

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[*] As proof sufficient of such failure, the single fact may be mentioned that the capacity of the left ventricle has been estimated as ranging between 1½ to 4 fluid ounces.

[†] Lancet, July 27, 1872.

[‡] "What to Observe at the Bedside." By the Lond. Med. Soc. of Observation, p. 109, Ed. 2. The spherical balls employed by T. B. Peacock will, at least in some conditions of the orifices, probably give even more precise results.

	Auriculo-Ventricular Orifices.		Arterial Orifices.	
	Left.		Left.	
	Males	Females.	Males.	Females.
Mean . .	$45\frac{17}{20}$	$41\frac{1}{3}$	$31\frac{15}{61}$	$28\frac{4}{5}$
	Right.		Right.	
Mean . .	$54\frac{12}{61}$	$48\frac{1}{4}$	$32\frac{21}{61}$	$30\frac{1}{60}$

Hence it appears that the mean excess in width of the right over the left auriculo-ventricular orifice equals about $8\frac{1}{2}$ lines in males, $6\frac{11}{12}$ lines in females: the pulmonary orifice exceeds the aortic in width by a mean amount in males of $1\frac{1}{10}$ line, in females of $1\frac{1}{3}$ line. And so, while the excess of width on the right side of the heart is relatively greater in the male at the auriculo-ventricular, it is relatively greater in the female at the arterial, orifice. In persons who have passed the age of fifty it would appear from Bizot's figures that the aortic orifice is wider than the pulmonary by a mean amount in males of 1 line,—in females of $\frac{1}{18}$ of a line. This deviation from the rule of earlier years is plausibly referred to the greater frequency of atheroma and calcification in the artery of the left side, whereby the resistant power of the vessel being diminished, it gradually undergoes dilatation.

(*c*) The circumference of an orifice being known, its *area* may be calculated by an easy formula. Herbert Davies, the first to make this calculation, which he bases on the actual width-measurements of Peacock,* gives the mean areas of the four orifices as follows : †

	Males.		Females.	
Tricuspid . .	1·78	Sq. in.	1· 6	Sq. in.
Pulmonary . .	1	,,	·97	,,
Mitral . .	1·27	,,	1·27	,,
Aortic . . .	·78	,,	·67	,,

As in the case of the right and left compartments of the heart, so in that of their orifices, it has been suggested that the differences, existing in circumference and hence in area, at death, are directly connected with, in truth traceable to, the manner of dissolution; and that in reality such differences either do not exist at all, or exist only to a very slight degree, during life. H. Davies, however, contends with convincing logic not only that the full amount of disparity found after death really exists, (and is necessary for the perfection of the circulation, during life,) but that the areas of the orifices are constant quantities, remaining unchanged during the systole and diastole of the ventricles.

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* Croonian Lectures, 1865.

† Proceedings of Royal Society, p. 267. 1870.

(*d*) "*Law of the relative magnitude of the areas of the four orifices.*"—Now it must be confessed that little of practical significance came of the knowledge of these orifice-measurements. The mere figures told little in respect either of the physiology or the pathology of the heart,—and as clinical observers went on accumulating and recording linear widths, they were often tempted to exclaim *cui bono!* Beyond the mere establishment of the amount either of widening or stenosis,—sufficiently obvious to the eye and palpable to the fingers,—nothing was gained by the troublesome process of measurement. But Herbert Davies[*] has succeeded in giving real interest to the naked facts by showing that a law regulates the respective sizes of the orifices in health, a law summarily expressible thus:

$$\frac{T}{M}=\frac{P}{A}$$

This law is disclosed on comparing the *ratios* of the areas of corresponding orifices. "Thus (adopting Peacock's absolute measurements):

$$\frac{\text{Area of tricuspid}}{\text{Area of mitral}}=\frac{1\cdot73}{1\cdot27}=1\cdot4 \text{ nearly.}$$

$$\frac{\text{Area of pulmonic}}{\text{Area of aortic}}=\frac{1\cdot0}{.78}=1\cdot3 \text{ nearly.}$$

Or, in other words, the area of the tricuspid appears to bear nearly the same relation to the area of the mitral, which the area of the pulmonic does to that of the aortic orifice; *i. e.*, were the tricuspid, for example, twice the size of the mitral orifice in area, the pulmonic would be twice the size of the aortic orifice in area, the two ratios only differing from each other by one-tenth." Further, the areas of any three orifices being known, that of the fourth can be calculated to within a very small fraction.

That some such law as this existed might have been, though it was not, long since divined. The reasons, why the proportions are such as they are, and none other, seem clearly established in H. Davies' acutely reasoned argument: as, however, they refer to the physiology, rather than the pathology, of the circulation, they need not be considered here.

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[*] Proceedings, Royal Society, 1870.

B.—IN DISEASE.

35. The diseases of the heart itself and its membranes of course variously affect, and moreover are in some cases mainly signified by changes in, the weights, capacities, linear measurements and areæ, to which we have just been referring. Such of these changes as are of practical consequence will be considered under the head of each individual disease.

36. But there are, besides, certain diathetic affections, in which the heart has no direct concern, and in which the bulk and measurements of the organ undergo very sensible *diminution*, as a consequence probably of the lessening mass and failing nutritiveness of the blood. To these conditions must, probably, be referred the deficiency in the mean weight and size of the heart of persons cut off by chronic phthisis and cancer, as demonstrated by Louis and Bizot. It may be that a phthisical individual, whose heart only reaches the par of health for his age and sex, is nevertheless clinically the subject of hypertrophy: his amount of heart is beyond the legitimate wants of his impoverished system.

The normal law of the areas of the openings seems to hold good in pure phthisis (H. Davies): this might be expected; as, the mass of the blood diminishing like the mass of the tissues, pulmonary obstruction, sufficient to enlarge the right areas notably, could scarcely be expected to arise. When obstruction of the pulmonary circulation of bronchitic mechanism exists, the right areas will gradually enlarge.

37. Again, *increase* of weight, bulk and outline are observed with striking frequency in those essentially diathetic states grouped under the title of "Bright's Disease." In cases of the sort, where there is no valvular impediment, and where the other acknowledged causes of compensatory hypertrophy are absent, the irritant qualities of the morbid blood (aided, it may be, by abnormal difficulty in the systemic capillary circulation [493, *b*]) probably excite the heart to sustained over-action, which in turn induces over-growth.

PART I.

CLINICAL PHYSICAL EXAMINATION OF THE HEART AND BLOOD-VESSELS.

§ I.—THE HEART.

38. The physical methods of examination applicable to the heart are: Inspection; Application of the hand and Palpation; Mensuration; Percussion and Auscultation.

SECTION I.—INSPECTION.

39. Inspection directs itself to: the form of the cardiac region; the condition of its integuments; and the visible impulse of the heart.

A.—IN HEALTH.

40. (*a*) *Form.*—In perfectly normal chests the part of the walls lying to the left of the middle line, and corresponding to the heart, does not differ perceptibly in form from that placed to its right; these two divisions of the thorax are symmetrical. But individuals, who have never suffered from pulmonary or cardiac disease, occasionally present a moderate excess of convexity of the cardiac region generally, as a result of natural conformation, of curvature of the spine, or of change of form produced by influences of a non-morbid kind. So, too, physiological depression, or flattening, of the lower part of the præcordial region, in particular, sometimes occurs. Woillez states that such depression, when really non-morbid, is observed in the corresponding right region also,—is in fact symmetrical; but, I confess, I cannot wholly agree with him on this point. Without questioning the general accuracy of his statement, I must observe that I have occasionally seen depression limited to the left infra mammary region, both in the male and

female, where very patient examination failed to elicit evidence of present or past disease, either of the heart, liver, lung, pleura, or diaphragm.

41. (*b*) *Integuments.*—The præcordial interspaces are of the same width, and lie on the same plane as their fellows on the opposite side; and the soft parts have the same characters on both.

42. (*c*) *Impulse.*—In the majority of healthy persons the impulse is visible only at the apex, which beats in the fifth interspace, and somewhat against the sixth rib, about midway between the vertical line of the nipple and the left border of the sternum; the area of visible impulse does not exceed a square inch. Various physiological acts modify the precise spot of visible impulse. Thus changes of posture elevate, depress, throw it upwards or backwards: inspiration lowers it somewhat, and by carrying the lung in front of the heart weakens its force; expiration has the converse effects. A full meal or flatulent distension of the abdomen raises the apex-beat somewhat, and throws it to the left; pregnancy has the same effect. As a rule, thin, tall persons have an impulse of greater visible extent than the short and stout: in the obese, none can be detected; in persons with short sternum, it can be seen in the epigastrium. Habitually, it is more extensive in males than in females, and in persons of nervous than of other temperaments.

To the eye the impulse seems gently heaving and gliding from above downwards and from right to left, of brief duration, free from abruptness, regular in rhythm, and single.

B.—IN DISEASE.

43. (*a*) *Form.*—The cardiac, or præcordial, region becomes arched forwards in the course of pericardial effusion. I have never observed this change of form in pericarditis previous to the occurrence of liquid effusion; but it may occur while the fluid is yet very small in quantity. The intercostal spaces widen,—their muscular planes eventually almost protruding beyond the level of the ribs; while the left border of the sternum is pushed more or less, never more than slightly, forwards: change of form so marked as this indicates abundant effusion, and may reach from the sixth to the second left cartilages inclusive.

Hypertrophy of the heart, especially of the left side, increases similarly the convexity of the cardiac region from the third to the seventh cartilages, and widens, but does not produce actual bulging of, the interspaces: pericardial adhesions, especially if associated with agglutination of the pericardium to the pleura and sternum, increase the prominence caused by any given amount of co-existent hypertrophy.

Solid accumulations in the lower part of the anterior mediastinum likewise arch the superjacent walls, and have occasionally caused obscurity in diagnosis, as well as errors in treatment [381, *b.*]

44. On the other hand, depression or excavation of the præcordial region may occur during the absorption-period of pericarditic effusion: commonly at the lower part of the region; occasionally, as I have once seen,[*] above its natural limits. In this instance the excavation formed during convalescence at the first and second left interspaces, close to the sternum. It has been the habit, on speculative grounds, to ascribe any such depression following pericarditis to the influence of adjacent pleurisy; but in point of fact, the change of shape sometimes occurs where all physical signs have argued against the existence of the latter inflammation.

45. (*b*) *Integuments.*—The integuments of the cardiac region are sometimes markedly more œdematous, than those of other parts of the chest, in cases of pericardial effusion lapsing into the chronic state. This condition, in itself unimportant, derives interest from its occasionally giving rise to sounds, audible with the stethescope, and simulating moist rhonchi and friction-sound.

46. (*c*) *Impulse.*—Disease alters the position, extent, force, character, and rhythm of the heart's visible impulse.

47. The apex-beat undergoes very marked changes in position in various diseases of the lungs, pleura, mediastinum, and abdominal organs;[†] but affections of the heart itself and of its external covering (with which we are alone concerned here) likewise displace it.

(*a*) All enlargements of the heart *depress* the apex-beat, and may carry it as low as the seventh interspace, or eighth rib: if both sides of the organ be equally affected, that point is commonly displaced to the left; if the right side be the main sufferer, the impulse is chiefly visible to the right, behind and below the sternum; where the left side is alone, or chiefly, diseased, the apex may be seen at variable distances from the natural spot, to about three inches and a half to the left of the nipple.* If, as is not very uncommon, hypertrophy affect, almost exclusively, the part of the left ventricle adjoining the mitral orifice, the base of the organ falls disproportionately to the rest, and the apex is thrown forwards. Procidentia of the apex in general hypertrophy is, *cæteris paribus*, greatest, where there is agglutination of the pericardial surfaces also. Aneurism of the arch of the aorta, or of the pulmonary artery, likewise lowers mechanically the point in question. It has been affirmed that atonic relaxation of the great vessels occurs in various acute adynamic diseases to sufficient amount to allow of an unappreciable fall in the heart's level: I have lately observed this in a case of typhoid fever.

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[*] Clinical Lecture, case of Craddock, "Lancet," vol. i. p. 144, 1849.

[†] Enlargement of the right, as well as of the left, lobe of the liver, may displace the heart's point upwards and outwards, and sometimes give useful and unexpected aid in diagnosis. Such displacement contributed much to distinguish acute abscess with enlargement of the liver from abscess of the abdominal walls, in a case which I published some time since. (Case of Fairbanks, Clin. Lect., "Lancet," loc. cit.)

(*b*) On the other hand, diminution of the size of the cavities, consequent on sudden enormous loss of blood, acting in conjunction with retraction of the great arterial trunks, slightly *raises* the apex-beat. It seems possible that a rude estimate of the degree of hæmorrhage might be formed through this changed position of the heart,—but of course only in individuals the precise point of whose normal beat was previously well known.

But the affection which most notably raises the apex-beat is pericardial effusion. When considerable accumulation exists in a pericardium free from old or recent adhesions, if the heart be not enlarged, and if there be no adjacent pleuritic adhesions, the increase of fluid pushes the base of the heart and the great vessels backwards and upwards, and twists the apex outwards and upwards, so that it beats opposite the fourth interspace or rib directly behind, or a little outside the nipple. The progress of elevation may be traced from day to day, as of the subsequent fall during the course of absorption. When the spot of the apex-beat is thus raised, it may always be *seen*, unless where general undulatory impulse tends to throw it into the shade: but it may or may not be *felt*; if not, the amount of effusion is in all probability very considerable. The possible sources of fallacy in interpreting this sign are fully considered with the subject of pericarditis. If during convalescence the apex gradually fall to below its natural site, hypertrophy, as an immediate sequela of the pericarditis, may be diagnosticated; for had the enlargement preceded the serous effusion, the apex would not have been raised.

I have further distinctly ascertained that where the pericardial surfaces are agglutinated together by recent and abundant exudation-matter, the interstitial contraction of this will sometimes suffice, unaided by fluid in the sac, to raise the apex-beat an intercostal space.[*]

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* The furthest point I remember to have seen is 3¾ inches outside the nipple (*vide* case of Hope, Clin. Lect., "Lancet," loc. cit., p. 415); this, too, is only possible in broad-chested persons.

(*c*) In the natural state of things the maximum of the heart's visible impulse corresponds to the apex of the organ; under peculiar circumstances that maximum may be transferred to the base. Thus in a case of highly-developed hypertrophy and dilatation of the left ventricle,[†] where numerous circumstances pointed to the probable existence of an aneurism of the thoracic aorta behind the base of the heart, the impulse was very notably greater about the third than the sixth interspace. As the case did not terminate fatally in the hospital, I had no means of positively ascertaining whether aneurism existed or not; but I do not believe that any affection of the heart's substance alone (except, perhaps, true saccular aneurism of the left ventricle), will thus transfer the maximum amount of visible impulse from the apex to the base. I refer here to purely systolic impulse.

(*d*) Instead of a forward movement accompanying the systole, a sinking inwards is sometimes to be seen. In some thin-chested people, with moderate hypertrophy, while the fifth interspace rises, the fourth sinks with the systole; the organ appears to go through a see-saw movement, and produces a momentary tendency to a vacuum at the fourth. So, too, the upper part of the epigastrium sinks in sometimes with the systole,—a sign originally deemed diagnostic of pericardial agglutination, and certainly, though more frequently absent, sometimes present in that state. I have known this systolic depression extend to the lower part of the sternum, and the two or three adjacent cartilages, in hypertrophy with agglutinated pericardium. Hypertrophy with dilatation will produce epigastric depression during the systole, in persons with short sternums, independently of adhesions.

(*e*) In some rare cases, the visible impulse appears horizontally double and pendulum-like.[‡]

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[*] Campion, U. C. H., Females, vol. vi., p. 39, 1852.

[†] F. Groove, U. C. H., Males, vol. viii., p. 13, April 25, 1849.

[‡] "Systolic action of heart, unnaturally visible in fifth interspace, from a finger's breadth to left of sternum to two fingers' breadth to left of nipple, or about three inches; movement distinctly pendulum-like,—a first blow appearing to be given at the outer part of space mentioned, a second at the inner; but by application of the finger it is found that the real impulse is only at the site of the external visible impulse. When the outer point is struck by the heart's apex, the inner point sinks in; when the apex recedes, the inner point recovers its natural position." Dunn, U. C. H., Males, vol. vii., p. 76; Feb. 17, 1852. The case was one of cancer of the stomach with great emaciation; no cardiac disease.

(*f*) The impulse may appear undulatory,—the wave-like motion, very variable in superficial area, may reach vertically from the first to the sixth interspace, and transversely from the left nipple to an inch to the right of the sternum. Its position may be altered, in the same directions as the heart itself, by changing the patient's posture. Commonly the axis of undulation is diagonal, from below upwards, and from left to right: undulation may be well marked in one interspace, imperfect or null in an adjoining one; in this case the line of current is generally horizontal.

When so fully developed that there can be no mistake about the fact, undulatory impulse is a very distinctive sign of fluid in the pericardium. But the sign unfortunately exists in a small proportion only of cases of hydropericarditis; and I do not remember to have ever satisfactorily observed it in mere dropsical hydropericardium. Again, a kind of pseudo-undulation is not an uncommon character of the impulse of weak and fatty, dilated hearts, especially where any physical conditions of the lung or pleura, combining with the dilated enlargement of the organ itself, tend to bring an abnormal extent of its anterior surface in contact with the chest walls.

Furthermore, undulatory impulse (unfortunately in regard of diagnosis) very perfect in kind, may exist in cases of pericardial agglutination, especially where this is combined with similarly close adhesive union of the præcordial pleural surfaces. Dilated hypertrophy being habitually co-existent, both systole and diastole seem impulsive;—dimpling inwards occurs with the systole at the apex, while a space higher a slight bulging may take place at the same instant: during diastole the converse conditions occur; and these alternating actions give a very distinct wave-like movement over the surface concerned. These actions may be more or less distinctly perceptible in four or even five spaces.*

(*g*) Some remaining perversions of impulse, being more readily substantiated with the aid of the hand than of the eye, are elsewhere considered. [54].

SECTION II.—APPLICATION OF THE HAND.

48. By application of the hand, the heart's impulse, the movements of the præcordial ribs and interspaces, and the state of vocal fremitus over the cardiac region are examined.

* Dell, U.C.H., Males, vol. xvii., p. 148. In infancy and early youth, the student must remember, this simulation of fluid movement may be to the last degree perfect and deceptive.

A.—IN HEALTH.

49. The visible movement corresponding to the heart's point is felt to possess a certain amount of impulsive force; which depends on the shock of the part immediately adjoining the apex of the organ against the side.

50. The essential physiological facts of clinical import, connected with this shock, are; (*a*) its production by both ventricles close to the point of the heart; (*b*), its synchronism with the systole of the ventricles; (*c*), its synchronism with the commencement of the first sound of the heart; (*d*), its anticipation by an almost imperceptible moment of the diastole of the neighbouring arteries.

(*a*) That the right ventricle plays any part in the impulse has been denied by Filhos, on the ground that the fibres of that ventricle, not being spirally arranged, are incapable of undergoing any other movement than those of contraction and dilatation. But, in the first place, there is no absolute certainty that the impulse depends on the action of the heart's spiral fibres at all; in the second, the phenomena of dilated hypertrophy of the right ventricle (which hypertrophy cannot be imagined to give a new direction to fibres) prove that powerful impulse may depend on that portion of the heart solely; and, in the third place, Reid, among others, has distinctly shown that the right ventricle does contain spiral fibres as well as the left.

(*b*) The systolic synchronism of the impulse, as commonly accredited, is not universally received. Taking the systolic coincidence as one theory, the total number of opinions concerning the rhythmic relationships of the shock amounts to four, the additional three standing as follow: first, the normal impulse synchronises with the diastole of the ventricles, and this alone (Descartes, Burdach, Beau, Cartwright, and others); secondly, the normal impulse is constantly double, systolic and diastolic (Verneuil,* Bellingham); thirdly, a diastolic and a systolic impulse both exist, but "succeed each other so rapidly in the normal state that they become, as it were, confounded together, and cannot be distinguished from each other." (Aran.) †

The majority of persons advocating these conflicting opinions appear to me to have embroiled this question, of which the clinical importance cannot easily be exaggerated, by mixing up with it the problem of the mechanism of the impulse. Assuming that a certain agency is the efficient cause of the shock, they infer that shock must be systolic, diastolic, or both, because the agency in question comes into force at one or other or both those periods of the heart's revolution. This is simply explaining a dubious fact by the aid of one yet more dubious—*ignotum per ignotius.*

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* Locomotion du Cœur, Thèses de Paris, 1852.

† Traduction de Skoda, p. 218, 1854.

Yet on the other hand clinical determination of the question is excessively difficult in the human subject; and the statical relationships of the contents of the thorax, as well as the conditions under which those contents normally move, are so gravely altered by taking away the wall of the chest, that the results of vivisections, even of large vertebrate animals, cannot be implicitly confided in. Although not unopen to objection, the observations made in cases of ectopia cordis in the human subject seem to supply the best materials for determining the time of the normal impulse. Now Cruveilhier,* Skoda,† and Mitchell,‡ who have each carefully observed a case of this kind, though they differ in many points, agree as to the synchronism of the impulse with the ventricular systole. And this result is satisfactorily confirmed by an observation made by Bamberger on a man, aged thirty, with a clean, gaping, penetrating chest-wound, below and somewhat anterior to the nipple at the lower edge of the fifth left rib. The finger introduced perpendicularly to the surface, encountered the smooth slippery apex: with every systole, the apex, hardened and brought somewhat to a point, glided on the finger, along the chest wall, in a direction from above downwards, and *somewhat towards the left*, passing down slightly beyond the inferior limit of the wound; whilst in diastole it was retracted upwards and became imperceptible.§ I accept, then, this synchronism as a demonstrated truth,—especially as the mass of observation of other kinds (*exempli gratiâ* that of the coincidence of the heart's first sound with the impulse), as likewise the weight of argument, deposes in its favour. That there is locomotion of the ventricles during their diastole, as a consequence of the influx of blood from the auricles, may be conceded; but there is a total want of evidence that in the healthy state such locomotion is productive of parietal impulse.

(*c*, *d*) The relationship in point of time of the ventricular systole to the first sound of the heart and to the diastole of the neighbouring arteries is considered with the account of the mechanism of the heart's sounds.

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* Gazette Méd. de Paris, Août, 1841.
† Perkussion, &c., 4e, Auflage.
‡ Dub. Journal Med. Science, 1844.
§ Quoted by Luschka, Op. cit.

51. *Clinical characters of the impulse, as felt.*—The impulse, slight in amount and imperceptible to the individual himself, mixed impulsive and gliding in character, free from abruptness or sharpness, yet decisive in rhythm, of brief duration, varies in force directly as the muscularity of the heart, and the energy and rapidity with which its fibres contract. However, in broad-chested and stout persons, though provided with powerful hearts, the impulse may be scarcely perceptible to the hand: it may then be detected by placing a stethoscope in the fifth interspace, and applying the ear as if for auscultation. In thin, tall persons, the impulse is disproportionably strong,—so also in women and children, in whom, too, it is distinguished by a certain abruptness and shortness.

Though, of course, essentially the same in position as the visible impulse, the shock may be felt a little lower than seen, namely, behind the sixth rib. This peculiarity is, however, not observable by any means in all people; it probably depends on an unusual amount of inspiratory descent of the heart during systole.

52. *Modifying influences.*—Forced expiration widens the area, and increases the apparent force of the impulse; inspiration affects both conversely. The varying condition of the neighbouring edges of the lungs at the two periods of each respiration fully explains this. Various acts quickening the heart's action intensify the shock—muscular exertion, walking at a rapid pace, and *à fortiori*, running, going against the wind, ascending heights, &c., have all this effect. Voluntary acceleration of the respiration will, especially in peculiar constitutions, act (indirectly, by quickening the circulation) similarly on the impulse; so too will the slight erethism of the circulation that accompanies digestion. But of all causes which act in this manner, independently of disease, moral influences and the passions are the most powerful; and among these, fear, perhaps, holds the highest place. The cardiac shock, under the agony of fear, may become so powerful, as not only to be distinctly perceptible to the individual, but to be actually and sharply painful. Simple "nervousness" acts, to a slight degree, similarly.

53. *Mechanism of the Impulse.*—It does not appear that, if the mechanism of the normal impulse be considered in itself alone, and without reference to the period of the heart's revolution at

which the shock is effected, any one rather than the rest of the numerous theories, adduced in its explanation, involves consequences of clinical significance. In various states of disease where the manner of the impulse might, if thoroughly understood, carry with it diagnostic weight, the mechanism indubitably differs from the normal type. It would consequently be unwise to devote any lengthened space in a purely clinical work to a discussion of the multitudinous speculations on the subject; a brief statement of the doctrine, apparently the most trustworthy, seems alone required.[*]

The mechanism is complex. (1.) During systole the ventricles shorten, and at the same time bulge in all directions, the bulging being most manifest a little above the apex. This bulging leads to centrifugal peripheric pressure in all directions,—among these towards the anterior wall of the chest: herein lies one component element of the impulse.[†] But only one element,—it is perfectly obvious that were this, as some persons maintain, the sole cause of the shock, a sensation of movement from below upwards would attend it, and not one, as is really the case, of movement from above downwards.

(2.) A swinging movement from right to left accompanies the systole. This can be seen distinctly, nay, even felt, in some thin-chested people; its reality was besides substantiated by Bamberger in the case of penetrating chest-wound already referred to. This movement is probably a resultant of the action of the spiral fibres of the ventricles, and is indubitably forcible enough to impress the chest-wall.

(3.) But there is yet another systolic movement, that of the total heart from above downwards, which plays its part. A number of observers testify to its existence; and as the ventricles shorten during systole, general downward movement seems a matter of necessity, otherwise, instead of impulse against, there must have ensued recedence from, the parietes: the systolic swelling of the substance of the ventricles obviously would not suffice to counterbalance the coëtaneous diminution of their length. Elongation of the great vessels is probably concerned in the systolic lowering of the heart's position.

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[*] As the "Segner's water-wheel," or recoil theory, ascribed to Gutbrod, and adopted with certain modifications by Skoda, has had many partizans, I think it right to observe that the combined objections of Valentin, Messerschmid, Kiwisch, and Aran seem to me to refute it victoriously.

[†] Various observers might be referred to in support of this change of form of the ventricles; the observations of Mitchell (loc. cit.) in his case of ectopia cordis are however enough for our purpose.

The normal impulse, then, seems to be a resultant of bulging of the ventricles, and of locomotion of the heart in two different directions, downwards and sidewards.* And the general laws of morbid action justify us in looking for varying degrees and combinations of these three elements of the impulse in disease.

B.—IN DISEASE.

54. I. *Impulse.*—(*a*) *In non-cardiac disease.*—In adynamic diseases, and in various non-febrile blood diseases, attended with depression of vital force, the heart's impulse habitually loses in strength.

Certain affections of the brain and spinal cord weaken the force of the heart's impulse; as do also various medicinal substances,—aconite, veratrum viride, tobacco, hydrocyanic acid.

Consolidation of the adjacent edges of the lungs, or abundant solid induration-matter in the contiguous pleura, solid substances in the mediastinum, and upward enlargement of the liver or spleen, lead to exaggeration of the heart's impulse; solid material being better fitted for conducting the heart's motion than spongy lung. Diminished size of the lungs, especially of the left, and pleuritic adhesions so placed as to withdraw the edges of the lungs from in front of the heart, or to cause by pressure atrophy of the enclosed pulmonary parenchyma, intensify the palpable shock for obvious reasons. The falling in of the chest-wall, which ensues on these conditions of the lungs and pleura, acts in the same manner. In all these cases, it will be observed, the exaggeration in force, and the extension of area are *surface-appearances*, and do not indicate any real increase of either on the part of the heart itself; many a supposed hypertrophy of the heart is nothing more than a simulation of the disease by some one of the physical conditions now referred to. On the other hand, emphysema and hypertrophy of the lungs, especially of the left, by bringing an unnatural amount of pulmonary tissue between the heart and the parietes, weaken the palpable impulse, and may completely mask the direct parietal shock of well-grown cardiac enlargement.

Again, the quantity and quality of circulating blood affect the heart's shock: at least the excited and sharply forcible impulse, occurring during reaction after hæmorrhage, appears in some part (perhaps frequency and force of propulsion in lieu of quantity propelled) due to the diminution of the blood at command. In the spanæmia of chlorosis, the impulse, though sharp rather than strong, is yet stronger than in health. The condition of the heart's substance does not explain this.

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* The weight of evidence seems to show that the alleged forward movement in systole is nothing more than anterior surface-pressure from swelling of the ventricles.

55. (*b*) *In cardiac disease.*—The diseased states of the heart which produce *real increase* in the force and area of impulse are,—morbid functional excitement (all the varieties of purely dynamic palpitation, angina pectoris, the paroxysm of "cardiac asthma," &c.), inflammation (cardiac and peri- or endo-cardial), and enlargement, especially if combined with adhesions of the pericardium. The influence of inflammation is merely functional and dynamic; increased impulse only exists at the outset of pericarditis, before effusion has occurred to any extent. The influence of enlargement, on the other hand, is organic and statical: pure hypertrophy increases to its maximum the force of impulse; hypertrophy with dilatation, the force and area combined; dilatation weakens force, extends area.

The impulse may be increased to such a degree as to shake forcibly the head placed on the stethoscope, nay, even to shake the entire body of the patient, and the bed on which he lies; and it may stretch diagonally from the eighth left rib to the first right interspace near the sternum, and transversely, in the third and fourth interspaces, from two inches to the right of that bone to as many outside the left nipple: and in such cases the impulse is very distinctly perceptible even in the left back. Between this, the maximum amount almost ever witnessed, and the natural state, all possible gradations are observable. Increased force, when extreme, is always combined with other modifications of impulse.

Increased impulse may be especially perceptible either behind the lower part of the sternum from the fourth to the seventh left cartilage, and at the epigastrium,—or below and about the left nipple, and between this and the left costal cartilages; in the former case the right ventricle is commonly most affected, in the latter the left. But exceptions to this rule occasionally occur; extreme relative hypertrophy of the right ventricle will, in addition to the impulse on its own proper side, produce so much to the left of the sternum, as to simulate that of enlargement of the left half of the organ.*

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* Kernis, ætat. 10, U. C. H., Females, vol. ii., p. 237, 1847.

If increased impulse be mainly traceable in a horizontal direction, the right ventricle is by some observers held to be the source of the increase, because enlargement on that side has a tendency to render the position of the heart more completely horizontal; whereas if mainly traceable in a vertical, or a vertico-diagonal direction, the left ventricle is at fault, inasmuch as its hypertrophy elongates the organ in those directions. I have occasionally found the former, frequently the latter, of these propositions correct; but neither is worthy of implicit clinical trust. Dilated hypertrophy of the left ventricle, when extensive, most certainly widens the impulse horizontally.

56. On the other hand, the force of impulse is *lessened* by effusions in the pericardium,—more readily in cases of passive than inflammatory accumulation, because in the former no excitement of the heart exists. Dilatation weakens the shock; fatty infiltration with softening has the same effect, although, on account of coëxistent enlargement, the positive amount of impulse be above the average of health; abundant deposition of fat under the cardiac pericardium generally enfeebles the impulse.

57. The *character* of the impulse varies as much as its force. The *apex-beat* may retain the natural impulsive and gliding character, and be merely increased in power. Or it may acquire a quick, abrupt sharpness; or convey the sensation of slow heaving, or pushing forwards against an obstacle: in hypertrophy of the left ventricle, the latter character is habitual; in dilatation, the former. Where weakened, the loss of power may be simple; or the shock be also abrupt and jerking, or marked by fluttering unsteadiness, as in various species of softening. The *general impulse* on either side of the heart may vary similarly.

58. *Rhythm.* (*a*) In rhythm, the heart's natural impulse, we have seen, is synchronous with the systole of the ventricles, and single. Now the impulse lags slightly behind the systolic sound of the heart, where fluid is accumulated in the pericardium, and the position of the apex is *not* elevated; before the impulse reaches the chest-wall, a stratum of fluid must be pushed aside. Besides in certain states of disease, the shock becomes double, the added impulse being systolic or diastolic. Double *systolic* impulse is an occasional, though rare, attendant on eccentric hypertrophy, a not uncommon one (it may be even treble, whence a peculiar jogging, shaking character) where, in addition to the hypertrophy, the pericardial surfaces are agglutinated, or covered with recent exudation

matter,*—more especially if the præcordial pleural surfaces be also adherent. Laennec noticed that in cases of great hypertrophy it seems "as though the heart swelled, and applied itself to the parietes at first by a single point, then by its whole surface, and finally sank back in a sudden manner." The sudden "sinking back" of the heart, Hope was the first to state, sometimes terminates in a jog or shock (back-stroke) obviously *diastolic* in time. He believed this "diastolic impulse" was strongest in dilated, though sometimes very considerable in simple, hypertrophy,—and imperceptible in simple dilatation: this accords with my experience also. The explanation of the phenomenon given by Hope, seems, however, scarcely admissible. "It is occasioned by the diastole of the ventricles, during which action the heart sinks back from the walls of the chest, and this sinking back terminates in a jog or shock, *occasioned by the refilling of the ventricles*, and constituting the diastolic impulse in question." But the mere expansion of dilated hypertrophous ventricles is doubtless in itself capable of conveying a forcible impulse to the surface; the intense power of even the natural diastolic expansion has always been insisted upon by vivisectors. On the other hand, there are many cases in which the diastolic sensation is rather one of *inward succussion* than *parietal impulsion;* it then depends, quite as probably, on falling back of the weighty heart against the spine,—a view strengthened by the fact that "diastolic impulse" is peculiarly obvious in cases where solid accumulations, aneurismal or other, lie behind an enlarged heart.† A *double* diastolic impulse has been described; this I have not observed,—it appears not improbable that impulses, so spoken of on the ground of their being unattended with arterial pulse, were in reality systolic, but too weak to reach the radial arteries.

(*b*) In the normal state, any successive number of impulses are equidistant, and of precisely identical, or very nearly identical, force; in diseased conditions of the organ this uniformity may disappear. There may be several strong shocks succeeded by others as feeble; and the number of each kind may be uniformly, or very nearly so, the same,—or their number may vary greatly,—in point of time all forms of irregularity are observable: force is rarely thus affected without time being implicated also. Simple hypertrophy of one or both ventricles does not produce this perversion of rhythm and force, unless some condition seriously affecting the pulmonary circulation occurs,—especially congestions, and inflammations of the lung. But both force and time are affected to a high degree, when dilated or simple hypertrophy of the left ventricle coëxists with mitral insufficiency; force more than time, where the same state of the ventricle attends aortic insufficiency. Marked dilatation, fatty infiltration, fatty metamorphosis, and mere flabby softness of the heart, produce great irregularities of rhythm and force of impulse,—but in these cases no single impulse is, absolutely speaking, powerful, unless the walls of the ventricles be thickened also. Pericarditis, both before and after the occurrence of liquid effusion, is occasionally attended with similar irregularity; so, too, are various malformations of the heart, especially during paroxysms of dyspnœa; here perversion of the relative capacities of the two sides of the organ probably acts as the immediate cause of the irregularity.

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* Courtenay, U. C. H., Females, vol. vii., p. 303 et seq.

† When a heart either quite healthy, or affected with enlargement so slight as to be practically insignificant, is excited by exercise or emotion, a slight jog synchronous with the diastole may often be felt. But this jog cannot, as it appears to me, be correctly spoken of, with W. Bellingham, as an impulse: the impulsive character seems to be wanting.

59. *Auricular Impulse.*—The impulses, whether natural or morbid, hitherto spoken of, are caused by ventricular action: does the contraction of the auricles produce visible or palpable impulse? Certainly not, in the state of health: their systole is feeble, and tends to withdraw them from the surface; their diastole yet feebler, passive and gradual in character. Where, however, the auricles are hypertrophous and dilated, it is conceivable that their systole, perhaps even their diastole, may become perceptibly impulsive. Auricular impulse, if systolic, should very slightly precede the shock of the heart's apex; if diastolic, it might coincide, in consequence of disturbed rhythm, with any moment of the ventricular systole, though, in all probability, it actually would coincide with the commencement of that act.

In a case where percussion-dulness at the second left interspace indicated an enlarged left auricle, impulse, preceding the ventricular systole, existed in the third left interspace close to the sternum; this was very probably auriculo-systolic. The left ventricle was hypertrophous, and the mitral valve insufficient.* It is probable, too, as suggested by P. Blakiston,† that the impulse of the ventricles may be communicated, in some cases of dilatation of the appendix of the left auricle, to the second left interspace:

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* Frederick Smith, U. C. H., Males, vol. v., p. 296, Oct. 1850.

† Diseases of the Chest, p. 124.

such *pseudo-auricular* impulse would synchronise with the ventricular systole.

60. II. *Form.*—The præcordial intercostal spaces are, in the natural state, of the same form and dimensions as the corresponding set on the other side of the chest. They become widened by hypertrophy,—widened, and even bulged outwards, by pericardial effusion, especially in the young; narrowed by chronic pleurisy and pericarditis, particularly if cardiac hypertrophy, sequential to the latter, have failed to occur.

61. III. *Movement.*—The state of movement in these spaces is better appreciated by the eye [47, *d*] than by the hand.

62. IV. *Vocal Fremitus.*—Where the heart is uncovered by lung, vocal fremitus is more or less completely annulled; the edges of the lungs at the cardiac region may hence be distinguished by means of this sign with a good deal of precision,—that of the right organ more satisfactorily than the left as a general rule.

Morbid alteration in the area of deficient vocal vibration over the heart-region is a sign scarcely appealed to in the diagnosis of cardiac affections. Perhaps it deserves to be more frequently employed. It cannot be questioned, for instance, that enlargements of the organ, especially of its right division, and accumulations of fluid in the pericardium will annul the fremitus to an unnatural extent to the right of the sternum. But the other signs of these states are numerous and positive; and, as far as I know, the only conditions under which this defect of fremitus might prove clinically valuable, would be where dull percussion-sound of undetermined nature existed at the right border of the heart. For, if this toneless percussion depended on induration of the lung, the vocal fremitus would be increased instead of being annulled or weakened.

63. *Adventitious phenomena.*—Application of the hand detects, under special circumstances, two kinds of phenomena which are purely adventitious—valvular thrill and pericardial friction-fremitus.

(*a*) *Valvular thrill*, or "purring tremor," resembles, in many cases, the vibration of the surface of a purring cat,—in others is more like the vocal fremitus felt over the larynx of persons with powerful bass voices. Varying in force in different cases and in different conditions of disease, it may change from moment to moment in the same person, according to the energy of the circulation,—mental or bodily excitement rendering it powerful, when, in the state of repose, it had been almost imperceptible. Habitually synchronous, and agreeing in rhythm, with the *ventricular systole*, it is felt mainly below and within the nipple, about the fourth interspace over an area of from one to three inches; or at mid-sternum on the level of the third rib, in the third left, and the second right interspaces close to that bone: if perceptible higher than these limits, and especially if so above the clavicle, this species of thrill ceases to be of purely cardiac origin. Habitually caused by forcible and rapid propulsion, in a rippling current, of blood—more particularly of blood altered in certain of its properties—through orifices narrowed and roughened by disease, it may in rare cases occur independently of textural change in the valves. When connected with valvular disease, it ceases, for obvious reasons, to be produced, unless a certain quantity of blood is propelled with a certain force through the diseased orifice; and hence, after existing to a high degree, it may disappear, because the narrowing which originally led to it has increased to an extreme amount, while the heart's force has, from some independent cause, more or less failed; a similar alteration, it is well known, occurs previously to death in certain organic murmurs of the heart.

The two *common* combinations of disease, in which cardiac valvular thrill is observed to the maximum degree, are insufficiency of the mitral valve with dilated hypertrophy of the left ventricle,—and constriction of the aortic orifice, coupled with hypertrophy of the same kind and seat: in the former case, the seat of the phenomenon is below and within the nipple; in the latter, at the aortic base. Now, the physical conditions of the phenomenon seem pretty fairly balanced in point of completeness in these two combinations; yet certainly cardiac thrill is more frequently met with as a dependance on mitral regurgitant than aortic constrictive disease. Hope supposed that this arose from aortic thrill being masked by the interposed sternum: but, although it is true, thrill, thus localised, may be sometimes rendered more distinct by causing the patient to lie on the left side, and so carrying the aortic orifice somewhat from its post-sternal position, I cannot help thinking the essential cause is a simpler one, namely, the less clinical frequency of well-defined aortic constrictive, than of mitral regurgitant disease. Be it remembered *clinical disease* is referred to, not mere *anatomical change*.[*]

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[*] T. K. Chambers' analysis of 367 cases of valvular affection, occurring in 2161 bodies, shows the aortic valves to have been affected (alone or in association with others) 257 times, the mitral only 248: but obviously this result and the affirmation in the text are in no wise incompatible (Br. and For. Med. Chir. Rev., 1853).

Basic systolic thrill is also felt in that *rare* affection, stenosis of the pulmonary orifice. In an instance of this kind, which recently fell under my notice (in a boy aged six) the thrill was sufficiently strong to convey a sense of titillation as far as the palmar end of the fingers.

Cardiac valvular thrill synchronous with the *ventricular diastole* is, as far as my observation goes, a phenomenon of rare occurrence. If this be true, aortic reflux, as well as tricuspid and mitral obstruction, seldom lead to its production. The force of the current is commonly too feeble for its generation. I have, however, met with a perfectly well-defined basic diastolic thrill in cases, where there was no anæmia, where all physical signs of aortic insufficiency were highly marked, and where, as it were, to complete the surety of the diagnosis, the patient's eventual death was of the instantaneous kind peculiar among valvular diseases to the defect in question. A loose vibratile portion of valve or vegetation, floating in the current, would doubtless greatly facilitate the production of thrill. This diastolic thrill may (the reverse of the attendant audible murmur) be better conducted upwards above the heart's base than downwards to the ensiform cartilage.

If mitral constriction be associated with very considerable hypertrophy of the left auricle, and if the surface of the valve be at all rough, diastolic (or, more accurately, præsystolic) thrill will, or may possibly, be felt.

I have never found acute endocarditis, where there was full certainty of the absence of old-standing valvular disease, attended with thrill. But I have occasionally known a faint systolic aortic thrill, never a mitral one, accompany palpitation in spanæmic women, and in persons who had suddenly lost a large quantity of blood. It has never been shown that nervous excitement of the heart, pure and uncomplicated, will produce thrill in people whose blood is healthy.

(*b*) *Pericardial tactile friction-fremitus*, like the analogous phenomenon of pleural fremitus, is considerably rarer, and, when occurring, of shorter duration, than audible friction-sound. It has much more of a rubbing character than valvular thrill, with which it may coëxist, and is more moveable than this, migrating from one part to another of the cardiac region within the course of a few hours, although the patient has retained, and is examined in, the same posture.

In doubtful cases pericardial may be distinguished from ordinary pleural fremitus by causing the patient to suspend his breath, for its rhythm is of course cardiac:* an excess of fremitus commonly attends the ventricular systole. Cardiac action may be supposed capable of producing pleural tactile fremitus, as it indubitably does audible pleural friction-sound; nevertheless, I do not remember ever to have observed the fact: the diagnosis would be very difficult.

A remarkable case has been recorded by T. Swett,† where a distinct "thrill" over the heart was caused by friction against an enlarged and "tuberculated" left lobe of the liver; the heart, as likewise the pericardium, proved "perfectly normal in all its parts." Cardiac action may then produce tactile fremitus beyond the pericardium, and even through the diaphragm.

(*c*) *Diagnosis of valvular thrill and pericardial fremitus.*—But, setting aside these exceptional modes of mechanism, and admitting that a given vibration, felt over the heart, is really produced either within the organ, or on its pericardial surface, is it possible to localise the phenomenon with confidence in one or other of these sites? In the majority of cases the diagnosis may be securely made by the intrinsic quality of the sensation itself,—in the one state a series of rubs, in the other a single, more or less prolonged, thrill. This mode of determination may, however, fail; numerous associated characters, very much like those available in the distinction of peri- and endocardial murmurs, will then afford help through the difficulty. Thus valvular thrill will be distinguished from pericardial fremitus by its apparent depth from the surface, its steadiness of force, its uniformity of position, its maximum force in the same site as that of valvular murmurs, its non-increase by bending the trunk forwards, its precise synchronism with the heart's systole, in rarer instances with its præsystole and diastole, and lastly by the coëxistence of valvular murmur. Characters more or less accurately the reverse attend pericardial fremitus.

SECTION III.—MENSURATION.

64. Mensuration of the surface corresponding to the heart, confirms inferences, otherwise deduced, as to the existence of certain affections of the organ; and sometimes becomes a useful auxiliary in diagnosis.

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* Such difficulty must, however, be rare: pleuritic fremitus produces the sensation of slow and deliberate, pericardial that of quick and abrupt, rubs.

† New York Journ. of Med., July, 1840, p. 6.

65. Thus, in health, the nipples are equidistant from the middle line; in enlargement of the heart and in pericardial effusion, the distance between the left nipple and that line undergoes increase, —to a very notable amount in some old-standing cases of dilated hypertrophy. The left nipple, too, is carried somewhat downwards by the same affections. Again, the space comprised between the upper border of the third, and lower border of the sixth ribs, an inch outside the sternum, is the same on both sides in healthy persons free from curvature of the spine: the diseases just named increase the measurement on the left side. The influence of the two affections is the same on these measures; it differs in regard of the following one. The vertical distance between the left clavicle and the heart's apex-beat is in health the same as between the right clavicle, and the point on the right side, lying on the same horizontal level as, and corresponding to, that beat; now, in pericardial effusion, the distance on the left side falls considerably below, in enlargement of the heart rises considerably above, that on the right side. This admeasurement is really useful in some cases of difficulty,—as, for instance, where an enlarged and dilated feeble heart beats with a quasi-undulatory impulse.

66. In forced inspiration, the expansion on the level of the sixth cartilage is in health slightly less on the left side than the right, a deficiency referrible to the influence of the heart. In pericardial effusion this deficiency, instead of, as might on first thought be supposed, increasing, actually disappears; the pressure of the fluid on the diaphragm throws an extra amount of work on the ribs, which move comparatively more than in health. When the fluid accumulates to a great amount, however, say twenty ounces, the præcordial ribs, also, move less than in health. I do not know how this matter stands previous to the occurrence of effusion in pericarditis; nor have I sufficiently examined the point, to say positively how the costal motion varies in the different varieties of enlargement of the heart, accompanied or not with valvular disease.

SECTION IV.—PERCUSSION.

A.—IN HEALTH.

67. Numerous difficulties stand in the way of accurate discrimination of the heart's outline by percussion. Above the organ lie blood-vessels of large calibre with their contents, forming a quasi-

solid mass; and, besides this, the variable amount of fat and areolar tissue occupying the mediastinum may prove a source of serious clinical difficulty. Again, the liver, furnishing a percussion-note but slightly differing in amount, resonance, and pitch of tone from its own, coasts the right inferior border of the organ. Furthermore in front of the heart are placed the sternum, costal cartilages and ribs, possessed of a special resonance. Add to all this, that the organ is in a state of perpetual restlessness, and constantly changing its precise axis, bulk, form, and mode of relationship to the surface, while the quantity of lung in front of it on either side is likewise undergoing the changes dependent on respiration. Yet, in despite of all these obstacles, it is possible by care to establish, with sufficient accuracy for all practical purposes, the position and bulk of the organ;—nay, to measure approximatively its thickness.

68. The heart cannot be percussed with success, unless the person examined be in the recumbent posture; and, except for the special purpose of ascertaining to what extent the organ (or the source of præcordial dulness, whatever it be) is moveable, not the slightest change of position, even of the limbs, ought to be permitted during the examination. The force used in striking will vary from the gentlest touch with the flat surface of the pulp of the middle finger, to a sharp tap with the ends of one or more fingers; it is a mistake to suppose that violent blows serve any useful purpose. The nature of the material, on which the patient reclines, exercises much less sensible influence on the result, than where the lungs are the subjects of examination.

69. Percussion of the chest, where the heart lies immediately beneath the surface, gives a short, slightly-toned, high-pitched sound; the parietal resistance is highly marked. These characters are modified very perceptibly, except in the intercostal spaces, by the special resonance of the sternum, ribs, and cartilages. The sound emitted by these textures is notably more prolonged, of greater resonance than, and of different pitch from, the cardiac; and when the heart happens to reach only just to the right edge of the sternum (so that the proper tonelessness of wet muscular substance and intra-cardiac blood cannot affect the percussion-results to the right of that bone in the right half of the chest), it may prove next to, if not absolutely, impossible to define with confidence the outline of the right ventricle. There is reason to believe that the texture of the sternum is in some persons peculiarly resonant; besides certain interfering acoustic conditions

(one of the most indubitable of which, *horizontal conduction*, will by-and-by be dwelt upon), tend to modify the general result.

70. Where the organ is invested in front by lung, the resonance partakes of course, more or less, of pulmonary quality, according to the thickness of the intervening stratum of pulmonary tissue. We may then clinically speak of the heart's *superficial* and *deep-seated* dulness.

(*a*) By reference to the diagram (page 3), it will be seen that the limits of the heart's *superficial dulness* must be as follows:—On the right a vertical line, extending at mid-sternum from the level of the fourth rib to that of the sixth; on the left an oblique line passing outwards and downwards at a more or less acute angle from the latter, opposite the fourth cartilage, and curving inwards again, somewhat within the site of the nipple, to the sixth rib, beside the heart's apex; inferiorly, a line gently sloping to the left, from the central point of the lower edge of the sternum, along the sixth cartilage. This is the extent of heart uncovered by lung in calm respiration; and the form of the part is obviously, though only rudely, triangular: the lengths of its sides have already been stated [12]. The area of the triangle is to be made out only by the gentlest taps with the pulp of a finger, either on the surface directly, or on a finger of the other hand, used as a pleximeter. The right border is with difficulty established, on account of the sternal resonance.

The outlines of this islet of superficial dulness vary with the conditions of the act of respiration. Forced inspiration diminishes the extent of its area; expiration increases that extent, especially in an upward direction and to the right; and by the kind of percussion described above, these changes in dimensions can without much difficulty be ascertained. They are, however, more frequently serviceable as tests of the freedom of play of the edges of the lungs, than of the condition of the heart.

The triangular shape of the dull-sounding space, it must be mentioned, not only undergoes change with the different periods of respiration, but is sometimes inclined to squareness in outline, and sometimes shapeless.

(*b*) Beyond the limits of superficial dulness, the outline of the heart may be ascertained by the deep-seated dulness elicited by firm percussion. The *deep-seated dulness* extends normally in a vertical direction, from the third to the edge of the sixth cartilage, and transversely from the left nipple to a little beyond the right edge of the sternum opposite the fourth cartilage; the longest measurement is the diagonal one, from the upper part of the third right cartilage (the right auricle) to the point of the apex-beat. In ascertaining these limits there are two main sources of perplexity. The first consists in the difficulty of defining exactly the line of the base; as the dulness, produced by the mass of large vessels there, is nearly as great as that of the heart itself: this perplexity, fortunately, only occurs where the edges of the lungs are unusually far apart. The second lies in the difficulty of separating the adjacent edges of the heart and liver. The pitch of the liver-sound is higher than that of the heart *in situ* (especially if *horizontal conduction* from tympanitic intestine does not modify the proper hepatic resonance); and in many cases ordinary percussion will succeed in eliciting a sensible fall in pitch as we pass diagonally from the liver to the heart. Sometimes a narrow space, say from a third to half an inch in width, may be detected, giving a slightly tubular note, and this space, easiest detected if the patient be made to keep his mouth open, while percussed, corresponds to the line of union of the two organs.*

The "auscultatory percussion" devised by Camman and Clark † certainly renders the change of pitch, on passing from the liver to the heart, more positive and definite than it appears under ordinary percussion, and may be called to our aid in puzzling cases. The method referred to consists in receiving percussion-sounds directly through a solid stethoscope to which the ear is applied, instead of, as in the ordinary way, receiving them weakened by diffusion through the air. By practice the process becomes manageable without the assistance of a second person; the observer, having placed his ear to the stethoscope over the lower part of the liver, percusses the surface diagonally upwards and to the left, in the direction of the heart, and is apprised of his reaching that organ, by abrupt change in the pitch of the sound. The original and suggestive paper of the American physicians is well worthy of study.

71. Certain physiological conditions modify the exact area of the heart's dulness. Its outline cannot be identical during the different periods of the heart's motor revolution: the chances that percussion gives the outline in the state of ventricular systole or diastole, may be roughly represented respectively by the numbers 4 and 7 [88, note]. The chief interest of this matter turns on the testing the correctness of results, obtained during life, by

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* I presume this is because at this line interruption of media takes place.

† New York Journ. of Med., July, 1840.

the actual size of the organ after death: it may be neglected in, and is in truth too delicate an enquiry for, ordinary clinical practice.[*] Again, the act of respiration, and alterations of posture of the trunk, will of course change the position of the heart's dulness. The area of superficial dulness is relatively less in infancy, greater in old age, unless the lungs become emphysematous, than in manhood.

72. And, further, the acoustic phenomenon, elsewhere designated as *horizontal conduction*,[†] may exercise a very notable and deceptive influence on the apparent area of the heart as defined by percussion. When moderately strong percussion is made, even on the central point of the heart-region, the impulse horizontally conveyed to the stomach, especially if that organ be pretty fully inflated, may elicit a response in the form of a somewhat tubular note mingling with the proper dull, toneless resonance of the cardiac substance.[‡] This horizontal conduction is not to be confounded with the well-known vertical conduction of the sound of a deeper-seated organ through another lying over it,—as when hepatic dulness mingles with pulmonary resonance in answer to percussion in the fifth right interspace. In the heart-cases referred to, we must first assure ourselves that the stomach does not lie behind, be it ever so deeply, the spot percussed; or if this surety be unattainable, we must percuss so gently as to make it impossible the blow could influence a deeply-seated organ.[§]

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[*] The heart is drawn up after death, both by the emptying of its cavities and by the expiratory collapse of the lungs; hence another of several reasons why slight differences may arise in clinical and post-mortem examinations. The mode of death, too, must influence the precise site of the heart.

[†] Diseases of the Lungs, ed. 4, p. 75.

[‡] Aubrey, U. C. H., Males, vol. xvii., p. 268.

[§] This horizontal conduction has an important bearing on the percussion-results of other organs. Aran, in his translation of Skoda (p. 409), criticises his author sharply for affirming that the "percussion-sound in the renal region may be completely dull when the kidneys are very small, and tympanitic when they are very large." Skoda proffers no explanation of the seeming paradox. Now I have had occasion to notice a case fully demonstrating the truth of the latter clause of Skoda's statement; to the other, Aran, of course, would not object. In a man with an enormous cancerous mass occupying the entire right half, and encroaching somewhat on the left half, of the abdomen, the right renal region gave amphoric resonance under percussion; and this circumstance, taken in conjunction with the facts that no intestine lay in front of the tumor, and that the urine was of normal characters, seemed to connect the mass with the liver. Yet the tumor proved to be wholly renal—the intestines were pushed in a body far away into the left abdomen; while, the right ureter being obliterated, healthy urine flowed from the healthy left kidney. And, further, the substance of the morbid mass lay in close contact with the abdominal lumbar wall in the very site of the amphoric resonance. Now, as no intestine lay between the tumor and the anterior abdominal wall, *the special amphoric intestinal tone must have been horizontally conducted through and along the vertebral column from the coils of bowel far away in the left half of the abdomen.*—Bowles, U. C. H., vol. xvii., p. 114.

B.—IN DISEASE.

I. *Non-Cardiac Disease.*

73. *Area of dulness enlarged.*—Various morbid conditions, independent of disease of the heart or its membranes, may *increase* the area of præcordial dulness. Among these may be enumerated pure atrophy of either lung, with diminished volume; consolidations of the portions of lung adjoining the heart; enlargement and elevation of either lobe of the liver; accumulations in the mediastinum or pleura; diminution of bulk of the left lung with lateral pleural adhesions, a state tending to bring an undue quantity of the heart into contact with the chest-wall; aneurism of the great vessels; and even, in infinitely rare cases, tumor, with constriction and pouching, of the œsophagus. In some of these affections it will be seen the increased area of dulness depends on the heart being abnormally exposed, in others on the addition of extrinsic dulness, dependent on some other structure. In all of them the actual physical state of the heart may be completely unaffected. Not so, when the extent of præcordial dulness is increased in consequence of the stagnation of blood in the right cardiac cavities, caused by obstruction of the pulmonary circulation in various forms of dyspnœa; here the heart's own dimensions are temporarily changed.

74. *Area of dulness decreased.*—There is only one thoracic affection independent of the heart itself which seriously *diminishes* the extent of præcordial dulness,—emphysema of the lungs,—particularly of the left, and especially when conjoined with bronchitis. The temporary influence of bronchitis in increasing the bulk of an emphysematous lung, and so masking the dulness of a very greatly enlarged heart, is well seen in cases, where, superadded bronchitis having yielded to treatment, the area of cardiac dulness undergoes sensible increase from the removal of pulmonary distension.* Conversely it may be seen that ascites diminishes the extent of the heart's dulness, by pushing the organ upwards under cover of the lung.

Venesection, as first shown by Piorry, will very sensibly diminish the extent of præcordial dulness, especially towards the right side, in persons whose right cavities had previously been loaded with blood. I can, from my own observations, vouch for this fact,—(rarely now-a-days to be substantiated, blood-letting is so seldom resorted to). A marked state of anæmia, by reducing the heart's distension, narrows the area of its dulness.

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* Case of Hope, Clin. Lect., "Lancet," loc. cit., p. 443, 1849.

II. *Cardiac disease.*

75. The area of præcordial dulness may be *diminished* or *increased* by disease of the heart itself.

76. *Area of dulness lessened.*—Diminution of the area attends primary concentric atrophy of the organ; but though decrease in bulk of the heart, and in calibre of the large vessels, often occurs to a very notable amount in the tuberculous and cancerous cachexiæ, it is seldom detected during life in either: adjacent pulmonary dulness renders its discovery difficult in the former case; it is rarely sought for in the latter.

In pneumo-pericardium the natural dulness disappears more or less completely in proportion to the quantity of gas accumulated; even if there be fluid, as well as gas, in the serous sac, the entire præcordial region may give more or less tympanitic resonance in dorsal recumbency. By changing the patient's posture from the back to the side, dull sound will be elicited in the then inferior, tympanitic sound in the then superior, part of the præcordial region: this interesting fact I succeeded in establishing in a singular case of traumatic communication between the œsophagus and pericardium.*

Area of dulness increased.—The cardiac affections which *widen* the area of præcordial dulness are materially more important than those just reviewed; they are referrible to three main heads: enlargement of the heart, and fluid accumulation or solid formations within or about the organ.

(*a*) Hypertrophy, in all its observed forms and sites, increases the extent of cardiac dulness: the position of the increase and the elasticity of the walls differ under different circumstances. Simple hypertrophy of all the cavities, or of both ventricles, or the same state combined with uniform dilatation, extends the outline of dulness downwards, and to both sides, more, however, to the left than the right: scarcely any impression is produced on the chest-resonance at the upper outline of the heart,—a proposition which applies, with scarcely an exception, to all varieties of true cardiac enlargement; for, as already mentioned, the tendency of increase of bulk is to carry the heart downwards. In general dilatation, simple, or combined with attenuation, extension of dulness is also observed; and very careful percussion will substantiate the existence of less parietal resistance in this than in the preceding cases. If enlargement be limited to either ventricle, the extension of the dulness takes place in the direction of the affected one; and what has been said concerning the site of impulse in such cases [55] applies to the position of dulness. Accumulation of fat under the pericardium, when sufficiently great to alter the limits of dulness, generally does so to the right side; for the simple reason that such accumulation begins with, and attains its maximum at, that side. The influence of fat in this manner is, however, very rarely to be substantiated. Hypertrophy of the left auricle carries deficiency of resonance into the second left interspace.

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* Case of Ramo Samee, U. C. H., in the ward of A. T. Thompson. The perforation had been produced in the attempt to swallow a long blunt instrument, a juggler's "knife;" the case terminated fatally, and the preparation is in U. C. Museum. —No. 3859.

If with the enlargement coëxist close pericardial adhesions, the extent of dulness is always proportionately increased, but more so to the left than the right side: this effect is still more perceptible if there be pleuritic agglutination in the left infra-mammary region. In some cases of old pericarditis the area of the heart's dulness is increased upwards: thus I have repeatedly found, in persons who had previously been under my care for rheumatic pericarditis, that more or less marked dulness existed, years after, in the second left interspace, even up to the second cartilage, and at the adjacent part of the sternum. This state of resonance may exist with or without obvious enlargement of the heart; if without such enlargement, it can only be explained by the presence of solid induration-matter, about the great vessels and base of the heart; if with such enlargement, it is explicable by the elevation, which the heart undergoes during the effusion-period of pericarditis, being maintained by agglutination of the pericardium, in spite of the depressing influence of the enlargement.

(*b*) Fluid accumulation in the heart's right cavities (of blood, of course), occurs to sufficient amount, under circumstances of obstructed cardiac circulation, to extend very perceptibly the area of dulness on the right of the sternum. The extension of dulness takes place mainly between the second and the fifth interspaces. The most important condition of the kind, practically, is dilatation of the right auricle and ventricle, combined with insufficiency of the tricuspid valve. But the accumulation of fluid and semi-solid blood in endocarditis sometimes considerably widens the area, both to the right and left; and where obstruction by fibrinous coagula is gradually effected in the pulmonary artery, a like gradual increase in the area of dulness to the right of the sternum, immediately dependent on consecutive clotting of blood in the right cavities, may clinically be established.

(*c*) Fluid accumulation in the pericardium, even when of small amount, and whether passively or actively dropsical, or the result of pericarditis, produces an extension of cardiac dulness. Four ounces of liquid will certainly widen the area of dulness,—perhaps even less than this will suffice. Clinical experience proves (the results of artificial distension of the pericardium obviously could not be accepted as conclusive evidence) that the pericardium undergoes distension most readily upwards, with greatest difficulty downwards, with medium facility forwards, backwards, and sidewards. When distended with fluid, the sac retains its original rudely pyramidal form,—the base below, the apex above. The level of that *base* at the front of the chest commonly corresponds to the lower border of the sixth rib, sometimes to the sixth interspace, in very rare instances to the seventh rib: in the latter class of cases the texture of the pericardium had probably undergone some chronic change, of a rarifying kind, before the occurrence of effusion—or at least been distensible with more than average facility by fluid pressure. In cases of extreme accumulation the diaphragm is arched downwards by the fluid; the epigastrium may thus be rendered somewhat prominent, and the dulness of the fluid is with difficulty distinguishable from that of swollen liver,—it does not reach sufficiently to the left side to modify the percussion in the hypochondrium. The *apex* of the pyramid, as the fluid increases, gradually rises to the second left cartilage, to the first, to the sterno-clavicular joint, and even to nearly an inch above the clavicle, displacing the apex of the lung at the inner aspect of the supra-clavicular region. As the fluid increases, it pushes aside the edges of the lungs, where they join in inspiration at mid-sternum, between the second and fourth ribs;* and this sideward detrusion, in cases of abundant effusion, condenses the adjacent edges of the lungs, and so increases the lateral extent of dulness. On the right side of the distended sac the state of vocal fremitus will sometimes guide to the line, where the condensed tissue and the fluid join,—strong over the former, feeble or annulled over the latter.

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* Bartlett, U. C. H., Males, vol. iv., p. 292. The lungs were *five* inches apart on the level of the second rib. Such extraordinary distension could only result from very slow progress of the disease: it appeared to have lasted in this case six weeks.

With equal superficial extent the dulness from pericardial fluid is more absolute, and the parietal resistance greater, than from hypertrophy; a fact probably depending on the more perfect approximation of the fluid, than of the solid, material to the walls. This distinction is too delicate to be trusted to at the bed-side; the dulness of effusion is better distinguished from that of general hypertrophy by its extensive range above the third rib, and its ordinary limitation to the sixth rib below; and from dilated hypertrophy of the right or left ventricle by not extending, disproportionately to the area of dulness, towards the right or left side. It must not be forgotten that an aneurismal sac, (we may suppose it filled with fibrine, pulseless, latent, and *pro tanto* deceptive,) of the transverse part of the arch of the aorta, and bulging inferiorly; or a small mediastinal tumor; or even superabundance of natural fat, placed just above the third left cartilage, and behind the sternum (a source of fallacy more frequent in persons with much subcutaneous fat than in the thin); may all three give to the dulness of a simply enlarged heart the triangular outline of that dependent on effusion in the serous sac. In all the instances referred to, the dulness of the heart itself possesses its own form, more or less closely that of a square or a parallelogram,—the relatively narrow and pointed addition above, caused by one or other of the three conditions mentioned, leading to the simulation of the triangular outline of fluid collection. And if an enlarged heart, with such an accidental appendage above it, be weak and flabby, and give a quasi-undulatory impulse, the distinction of the case from one of pericardial effusion, especially if there be acute pyrexial symptoms and the commemorative history be imperfect, becomes one of most serious difficulty.

(*d*) The plastic exudation-matter of pericarditis sometimes forms a layer, one-third to three-fourths of an inch thick, of solid substance applied to the heart's surface: if chance place this great thickness of substance on the lateral confines of the organ, the area of dulness must be proportionately increased; but the sign is one of too great delicacy to be clinically serviceable.

(*e*) Cancerous accumulation in, or underneath, the pericardium affects the præcordial resonance in proportion to its amount.

78. *Quality of cardiac percussion-tone.*—The *quality* of the percussion-sound undergoes no very material change under the circumstances hitherto referred to, except in cases of pneumopericardium. In this affection the tone becomes tubular, amphoric, or genuinely tympanitic. Further, in those very rare

morbid states, calcified induration of the heart's substance and pseudo-ossification of the pericardium (of neither of which have I had much clinical experience), the resonance would, theoretically, become more ringing and osteal in quality than natural, and at the same time the resistance of the parietes prove sharp and highly marked [540].

79. *Altered outline of cardiac dulness.*—The *form* of *deep-seated* dulness, peculiarly affected, as we have seen, by pericardial effusion, remains essentially unchanged by general hypertrophy; excessive hypertrophy of any particular part must of necessity modify the character of the general outline,—but rarely to an amount that can be defined at the bed-side. Dilatation renders the shape more or less markedly square. Again, hypertrophy alters the form of the *superficial* dulness by pushing aside the lungs, and converts the small triangular, into a relatively large and irregularly square, space. Solid masses under the pericardium change the outline of dulness according to the direction of their growth; but their influence is very slight in this way, and on their rarity it is needless to insist.

Valvular diseases exercise no direct influence on præcordial resonance; the increase of dulness so frequently coëxisting with them of course really depends on some form of attendant enlargement of the heart. The membranous inflammations, when acute, though probably slightly increasing the heart's bulk by congesting its substance, do not *per se* (endocarditis at any period, pericarditis until exudation has occurred,) appreciably alter præcordial dulness.

80. *Moveableness of cardiac dulness.*—Præcordial dulness, whether dependent on fluid in the pericardium or enlargement of the heart, moves from side to side somewhat, as the posture of the patient is changed. Old pericardial adhesions, even, will not prevent sideward locomotion of a hypertrophous heart,—nor will pleuritic adhesions, in front of the heart in addition, do so, unless they be at once closely agglutinative and extensive.

SECTION V.—AUSCULTATION.

81. Auscultation, directed to the heart, analyses:—(I.) Certain sounds produced by the normal action of the organ; (II.) Modifications of these occurring independently of heart-disease; (III.) Morbid states dependent on heart-disease. Besides (IV.) the state of the respiration-sounds and (V.) that of vocal resonance,

specially in the præcordial region, sometimes afford useful information.

82. While the heart is ausculted, the patient should, as a rule, be placed in the recumbent posture, with the head slightly raised; unless, from the nature of his disease, this posture be an uncomfortable one. In as much as the precision of the notions, obtained from the examination, depends greatly on our being able to connect the spots, where various sounds are heard, with certain parts of the heart itself or great vessels, it is obvious that, in order to both simplify our task, and give all attainable surety to the indications, we should habitually auscult patients in the posture in which clinical practice must present them to us most frequently.

83. If there be any doubt about the superiority of mediate or immediate auscultation, in the case of the lungs, there is none in that of the heart. Certain phenomena, well audible at a given point with the stethoscope, may cease to be perceptible *one-third of an inch* beyond that point: such limitation as this, which is by no means very uncommon, evidently could not be substantiated by direct application of the ear to the surface.

84. The heart's sounds may be more or less masked by certain morbid states of the respiration-sounds,—and they may be given unnatural characters by the chance coincidence of the sound of natural inspiration or expiration; hence the necessity of causing the patient to hold his breath from time to time, while the heart is ausculted.

(I.) NORMAL CARDIAC SOUNDS.

85. Each complete revolution of the heart is accompanied by two successive sounds, audible by means of mediate or immediate auscultation in the præcordial region, and separated from each other by intervals of silence. These two sounds differ in all their characters,—intensity, duration, mode of propagation, pitch and quality: and the two periods of silence differ in the only character in which they can differ, namely, duration. These differences are found, provided the posture of the patient and the position of the stethoscope be unchanged, to be maintained either without the smallest, or with very slight, variation in any series of beats of the same heart.

86. The first of these two sounds, coincident with the commencement of the systole of the ventricles, the closure of the mitral valve, the heart's shock against the side, and the pulse, or diastole, of the arteries next adjoining the heart, is called the

first, or systolic, or (because typically of maximum force at the lower part of the cardiac region) [97], inferior, sound of the heart.[*] The second of the two sounds, synchronous with the diastole of the ventricles, the recedence of the heart from the side, and the pulseless state, or systole, of the large arteries, is known as the second, diastolic, or (because of maximum loudness at the upper part of the cardiac region) superior, sound of the heart.

87. The noiseless period succeeding to the first sound may be called the first, or post-systolic silence ; that succeeding the second sound, the second, or post-diastolic silence.

88. If the period of an entire revolution of the heart, that is from the commencement of one first sound to the commencement of the next succeeding first sound, be divided into ten equal parts, the durations of the several periods of sound and silence will be found on an average very closely as follows :—

First Sound . . . . = ·4
First Silence . . . . . = ·1
Second Sound . . . . = ·2
Second Silence . . . = ·3

The total duration of sound would hence stand to that of silence in the ratio as 3 : 2. This estimate is based on the results obtained by careful auscultation of a series of healthy males : it is only to be taken as approximately true,—both because there are very sensible differences in individuals, and because error is easily committed in estimating the extremely minute divisions of time concerned. Thus, when the pulse beats as much as from eighty to ninety times in the minute, the post-systolic silence is difficult enough of detection ; but it becomes obvious, where the pulse does not exceed fifty to sixty in a minute. The estimate, also, refers only, the student will remember, to the periods of *sound* and *silence*, not to those of *action* and *inaction*, of the heart : clinically there is no trustworthy plan of measuring the length of the ventricular systole and diastole.[†]

Any very notable deviation from the healthy standard is easily enough appreciated by the ear; as, for instance, in certain cases of dilatation, where shortening of the first sound and lengthening of the first silence rarely fail to arrest attention.

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[*] The instances are, however, by no means exceedingly rare, in which the first sound is louder at the basic than the apex region of the heart.

[†] It is obviously an error to maintain that the first sound and ventricular contraction are of equal length : as matter of theory, the contraction may be very much shorter,—the conditions giving rise to sound ceasing a more or less long time before the sound itself ceases. Recent enquiry (Burdon Sanderson, Chauveau, and others) shows that the duration of ventricular systole is notably less than that of diastole,—in the ratio 4·7 according to Morant Baker (Kirkes's Physiology). [210]

89. *Characters of the heart's sounds.*—An accurate idea of the character of the heart's sounds cannot be given by a single description of them, as heard in any one particular spot: they vary materially at different parts of the cardiac region, both in their positive and relative properties. The least study of the healthy chest will convince the student that the description which it is the habit to apply to the sounds of the heart generally, holds good only when these are heard towards the left apex. The sounds require comparative analysis: (*a*) at both sides of the apex-region, and at both sides of the base-region; (*b*) at base and apex on the same sides of the organ; (*c*) at base and apex on opposite sides. Now this is the most difficult of all studies connected with the healthy heart; but it is essential as the basis of observation of the organ in a state of disease.

(*a*) *At the left apex* the first sound is dull, measured, booming, prolonged, and strongly accentuated; its commencement pretty sharply defined, its close much less so. Double the length of the second sound, of lower pitch than this, and seeming to the ear deep-seated, it attains its maximum at this apex, in regard of accentuation, prolongation, and measured, booming character, *but not necessarily in amount of sonorousness.* The second sound, only half as long as the first—clear, abrupt, shock-like, and short—is more sonorous, more superficial, and of higher pitch than its predecessor.

At the right apex-region, the first sound is considerably clearer, shorter, more abrupt, less strongly accentuated, and of somewhat higher pitch than at the left apex-region. This difference of character depends, probably, both on the thinness of the walls of the right, as compared with the left ventricle, and on the parietes being of different conducting power in the two situations: at all events the sound, audible at the end of the sternum, may be fairly referred more particularly to the right ventricle; that near the nipple, to the left ventricle. So, too, the second sound is habitually clearer, and sometimes even of higher pitch, at the end of the sternum, than towards the left apex; this is, however, less constantly true, and I have known the converse to be the case, where no suspicion arose of the existence of dilatation of the left ventricle—where, indeed, the first sound possessed to the full its natural share of dull, prolonged, booming character.

Passing from the apex *to the base*, the same kind of dissimilitude, laterally, in the sounds, may be detected,—not indeed precisely at the base, but a little above this, opposite the second interspace, where the aorta and pulmonary artery lie in contact. The first sound at the right second interspace is commonly duller, of slightly lower pitch, and more prolonged than at the left corresponding point: in neither is it accentuated. The second sounds differ here in the same characters and in the same manner, but to a less degree: they are accentuated in both places, more on the right side than the left.

(*b*) Let us now examine *the two sounds comparatively at base and apex on the same sides of the heart.* The first sound, strongly accentuated at the left apex,—prolonged, booming, and dull,—at the left base loses the accent, which passes to the second sound, while this becomes louder, more ringing, and sometimes even of higher pitch than at that apex. At apex and base on the right side, the characters of the first sound are very similar; it has, however, more accent in the former than in the latter spot, both positively, and *à fortiori* as compared with the second sound.

(*c*) The relative characters of the *two sounds at base and apex at opposite sides of the heart* (crucially taken, as it were,) may easily be deduced from the foregoing account.

90. *Articulate symbols of the heart's sounds.*—Now, if these statements be correct, it appears obvious that no single articulate symbol can be devised, applicable to the heart's sounds in more than one point. The subjoined series is offered for the four points of prominent clinical interest. The acute accent-mark is used to show where the accent falls,—twice, when it falls very strongly; the marks of long and short, where length or shortness of sound is a prominent characteristic.

	First sound.	Second sound.
At the left apex	oū̋bb	dŭp
At the right apex	úp	tŭp
At the left base*	up	tŭ́p
At the right base*	ub	tŭ̋pp

The eye gathers from these symbols the tendency to equalization in length observable in the sounds at the bases, as well as the transference of the accent from the first to the second sound at the apices and bases respectively.

It may not be irrelevant to mark that these symbolic differences would suffice in themselves to show that *each side of the heart plays its separate part in generating the cardiac sounds,*—that the two sounds heard at any distance from the heart are in reality each of them compounded of two.

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* The left and right second interspaces close to the edge of the sternum.

91. *Propagation of heart's sounds, superficial and linear.*—(*a*) The extent, to which the heart's sounds are audible in health, is not subject to any fixed rule.* One great mistake, commonly committed by authors who attempt to define it, consists in their omitting to consider *separately* the first and second sounds. From this omission, the ordinary starting proposition, that "the heart's sounds are at their maximum in the præcordial region," becomes an error: the second sound is, in truth, heard in nine people out of ten more loudly at mid-sternum, on the level of the second interspace, than at any point of the præcordial region,—even limiting that region to the space in which the heart is uncovered by the lung during tranquil breathing. The thickness of the soft parts, the form of the chest, and many other physical conditions perfectly independent of disease of any of the thoracic organs, modify the extent of propagation so variously, that there can be no practical utility in laying down rules subject to perpetual exceptions.

(*b*) But the lines of propagation of the two sounds severally agree in most healthy persons, whatever be their absolute intensity at their seat of production; changes in these lines point positively to some modifying cause, and hence their establishment is clinically valuable. Now, the first sound passes slantingly upwards to the left acromial angle, growing weaker and weaker on the way; it loses much more on the route to, and at, the right acromial angle: its propagation backwards is clearest and fullest to the left,—so that while audible at the left back, it may be inaudible at the right. The second sound, with the base-region as its centre, radiates to the right and left acromial angles, with greater clearness to the left than the right; posteriorly it reaches the surface at the right side less clearly than at the left.

92. *Mechanism of the heart's sounds.*—The difficulty of unravelling the mechanism of the normal sounds of the heart is broadly and emphatically proved by the fact that, from the time of Laennec to the present day, some five and thirty theories, more or less completely differing from each other, have been proposed in its explanation. The subject seems at once a tempting and an

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* Some authors speak of the sounds being audible in healthy male adults, of moderate stoutness, even *at the right side posteriorly;* others write that the space over which they are heard *seldom exceeds two square inches in the præcordial region!* The truth is that either of these states may exist in different individuals in perfect cardiac health: the error lies in regarding either of them as constant.

exhaustless one. The zeal of experimentalists, unsatisfied with any one of the long series of conjectures previously proposed, seeks constantly to add something novel, either in the way of fundamental principle, or of combination of agencies already acknowledged or suggested, to the mass of acquired hypotheses. This zeal is commendable,—and would be above all praise, were it not too often associated on the part of fresh investigators with a disposition to sneer at the attempts of predecessors as capable and as conscientious as themselves, to ignore the perplexity of the problem, and to regard with overweening confidence the result of their own inquiries. One scarcely ever stumbles on a new effort to remove the mystery hanging over this part of the heart's physiology, without having the memorable words of Turgot vividly recalled to the mind:—" Moins on sait, moins on doute; moins on a découvert, moins on voit ce qui reste à découvrir . . . . Quand les hommes sont ignorans, il est aisé de tout savoir."

Were we to address the mass of practical physicians, at home and abroad, who are untrammelled by the instincts of paternity in regard of any of the theories proposed, and who have besides directed serious attention to the study of heart-diseases, and ask them—does any single explanation of the cardiac sounds heretofore adduced appear to you to solve the problem?—the all but unanimous reply would, there can scarcely be a shadow of a doubt, be—No. For this reason, I think, it would prove a useless, as it most certainly would be a tiresome, task to review the group of theories *seriatim*. The better plan seems to be to enumerate and describe, with as much brevity as possible, the dynamic phenomena, which are both coincident with the sounds and also by possibility soniferous, and endeavour to ascertain, on such acoustic, anatomical, experimental, and clinical data, as we can command, which among the number actually concur in producing the sounds.

93. *Mechanism of the first sound.*—The phenomena which are at once conceivably soniferous and coincident with the heart's first sound may be arranged in the following manner.

(*a*) *At and immediately after the commencement of the sound.*—At this period occur (1) the sudden tension of the auriculo-ventricular valves; the sharp collision of certain portions of the auricular surfaces of those valves; the impulsion of some of the blood in the ventricles against the ventricular surface of those valves: (2) The attrition of the blood-elements *inter se* within the ventricles, and their impulsion with attrition upon and among certain portions of the columnæ carnæ: (3) The impingement of the blood, projected from the ventricles, against the orifices of the pulmonary artery and aorta: (4) The forcible shock of the ventricular, against the bases of the aortic and pulmonary columns of, blood: (5) The simultaneous flattening of the sygmoid valves against the arterial walls; (6) the diastolic tense extension of those walls: (7) The impulse of the heart's apex against the side, or against lung-substance, if this be interposed: and (8) The attrition of the pericardial surfaces accompanying systolic movement.

(*b*) *Towards the close of the sound.*—The collision of the surfaces of the ventricles, after the expulsion of the main mass of their contained blood.

(*c*) *Throughout the entire duration* of the sound, the sustained muscular contraction of the walls of the ventricles, together with, as a specific additional element of sonorousness, the perfect, completed tension of the muscular fibres.

Before considering the claims of these different agencies as productive of the first sound, let me remind the student of a very obvious truth commonly ignored or forgotten by writers on this question,—namely, that sound or noise may by possibility be caused at the period of systole by many actions which, nevertheless, take no part in producing *the* first sound. It might be, again, that such superadded actions produced sounds, which sounds were habitually audible at the præcordial surface, or, on the contrary, either constantly or occasionally drowned *in transitu* thereto, either by the superior intensity of the true first sound itself or by interfering vibrations.

94. This premised, let us ask, first, which of all these possible elements of sonorousness actually prove soniferous,—and, secondly, which of them contribute to form *the* first sound of the heart. The order in which we examine them is not of much importance.

(*a*) The amount of noise, produced by the impulsion of the heart against the chest and its friction against the parietal pericardium during systolic locomotion, is too slight in the normal state of things to be audible to the listener through the chest-wall. Let, however, the action of the heart be simply abnormally increased in intensity, all other conditions remaining the same, and both these phenomena become perceptibly sonorous, each in its special way,—the former as a knocking and somewhat toned noise against the side, the latter as a rubbing murmur. Both these facts are almost daily exemplified in clinical practice: patients with hearts excited by simple neurosis, or with hearts somewhat enlarged and dynamically excited besides, very frequently present a pericardial rub of single systolic rhythm which disappears under rest and treatment.[*] And even where there is no statical or functional disturbance of the heart, a knocking sound may in some individuals be heard with systole, especially where the posture of the trunk, the period of the respiratory act, or other conditions, extrinsic to the heart itself, allow the surface of the organ to play with unwonted vigour against the parietes. Still the practised ear may always succeed in recognising that this sound is not *the* sound of systole,—but that it is a something superadded and independent, differing in site, in quality, and sometimes in precise rhythm from this.[†]

(β) There is a deficiency of evidence to show, that under the peculiar conditions of ordinary systolic contraction, the collision within the ventricles of the blood-elements among themselves is habitually soniferous; and, at all events, it may be assumed that any noise thus produceible fails to be conveyed to the outer surface of the chest.

(γ) Nor do I think proof has ever been given that the collision of the internal surfaces of the ventricles is attended with appreciable noise,—much less that such collision normally contributes to the formation of the systolic sound at its close. The hypothesis seems, in truth, confuted at once by the fact that the blood of the ventricles is not completely expelled by their systole.[‡] It has been suggested that a clicking quality in the first sound, sometimes to be detected after profuse hæmorrhage, may thus be generated: the idea seems by far more fanciful than judicious,—and, even conceding its justness, it throws no light on the mechanism of the normal first sound.

(δ) That the systolic sound derives its special dull, booming quality, and, to a great extent, its prolongation, from the act of muscular contraction, has since the day of Laennec been habitually accepted as a demonstrated fact. For, it is urged, on the one hand, the sound has been heard to retain these characters, *when the heart contracts after separation from the body and the action of the auricular valves is prevented*;[§] and, on the other hand, a very close imitation of the systolic sound is to be found in that of a powerful voluntary muscle abruptly contracting. I may mention that in a case, unique as far as my experience and reading extend (seen with Mr. Hardwicke), of hypertrophy of the recti abdominis, attended with powerful contractions, partly reflex, partly consensual, of their tissue, the variation in intensity of the muscular sound, according as the contractions were slow and gradual, or quick and abrupt, was very striking: in the latter case, the sound bore, to my ear, a very distinct resemblance to the first sound of the heart. But, on the other hand, similarity of two given sounds must not be taken as evidence that their mechanism is identical, or even allied, in nature. Thus a very fair imitation of the systolic sound may be made by snapping together firmly, but not too firmly, the ball of the right thumb and middle finger close to the ear. Yet there is not to be found among the wildest speculations on the cause of the first sound any hypothesis of mechanism assimilable to this.

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[*] *e. g.* Roberts, U. C. H., Females, Oct. 10, 1850.

[†] Where a thick layer of emphysematous lung intervenes between the heart and chest-wall, and sometimes even without this, I think there is reason to believe the first sound may be given an *intermittently murmur-like character* at the apex, independently of any disease in the mitral valve, simply by the apex-point of the heart impinging against the lung, and moving some of its contained air. I have observed the phenomenon (even during temporary suspension of the respiration), where this seemed its sole plausible explanation.

[‡] As is well known, blood may still be *seen* in the interior of the ventricle of the frog after the most complete systolic expulsion possible in the organ.

[§] This is difficult to conceive, and the statement is certainly at variance with

(*e*) The abrupt vibration of the stretched auricular valves has been held by many to be the sole, by others to be an important, element in the production of the sound. The latter class of reasoners argue that this membranous vibration impresses the special twanging character on the sound,—a character which may be detected by attention at its outset, and which, in certain states of altered contraction of the muscular fibres, almost covers or rivals in strength the sound generated by these: the elements of sonorousness of individual quality exist in both these actions; and, when either is experimentally interfered with, a corresponding change is found to follow in the character of the first sound. Halford, exposing the heart of a living animal and cutting off its supply of blood by pressure on the afferent veins, drew the conclusion that the sounds ceased then to be heard, because there was no blood to act on the valves. This experimental result is, however, capable of a very different interpretation, as we shall by-and-by see; and that valvular vibration can produce any sound is denied by A. Leared, on the ground that an amount of strain on the part of the valves, *immersed as they are* in a viscid fluid, would be required for the purpose, which cannot be conceived to occur.*

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recent and careful experiments, in which the mere cutting off of the blood-supply has been found to put an end to the sounds.

* On the Sounds caused by the Circulation of the Blood. London, 1861. Malherbe (Journal de Physiol. de Brown-Séquard, t. i.), also maintains the auriculo-ventricular valves cannot produce any sound at all.

(ζ) It seems possible the sharp shock of some of the blood against the ventricular surfaces of the valves plays a part in generating some noise of systolic rhythm; but with *the* systolic sound it has probably little or no connection, even as a reinforcing agency.

(η) It has been maintained, particularly I believe by Hope, that the walls of the ventricles become at a certain period of systole at once tense and vibrating, after the fashion of a stretched membrane, and so produce a specific addition to the muscular sound of contraction. This theory (wholly unsupported by experimental evidence) seems, acoustically as well as physiologically, of questionable justness, and may with safety be ignored.

(θ) That the projection of the ventricular blood against the orifices of the large vessels, and against their flattened sygmoid valves, combined with the diastolic expansion of the vessels, is productive of some shock-sound, seems scarcely questionable; for a sound is audible in the arteries synchronously with the heart's systole, under circumstances in which the idea of mere conduction from the heart is quite inadmissible. Such sound may be heard in the femoral and even popliteal arteries sometimes, where no disease of these vessels or of the aorta exists: in a word, the diastole of the arteries is in itself sonorous.

(ι) The collision of blood with blood is, according to A. Leared (*loc. cit*), the real cause of the first sound. Neil Arnott had forty years ago laid the groundwork of a blood-concussion theory in the following words:—

> "While water is running through a pipe, if a cock at the extremity be suddenly shut, a shock and noise are produced there. The reason is, that, the forward motion of the whole water contained in the pipe having been instantly arrested, and the momentum of a liquid being as great as of a solid, the water strikes the cock with the same force as a bar of metal, or a rod of wood having the same weight, and moving with the same velocity. A leaden pipe, if of great length, is often widened or burst in this experiment . . . .
>
> "The circumstances attending the circulation of the blood through the heart will, on consideration, be found to present every condition necessary for the application and illustration of this law; there is the current of blood passing through tubes, and this current suddenly and forcibly arrested by the closure of the valves."

A. Leared, working independently, concludes, from experiments and close reasoning, that the sole cause of the first sound is the forcible impact between fluid in motion (that coming from the ventricles), and fluid in a state of momentary rest (the blood columns in the aorta and pulmonary artery). The experiment of Halford, just referred to, is naturally appealed to in confirmation of his view by A. Leared: the true element of soniferousness, blood-shock, cannot be present, when there is no blood to produce the shock.

The contingent conditions to which A. Leared has, on proven grounds, attached importance, are: sufficient viscidity of the blood; sufficient force in the circulation; sufficient pressure upon the blood; the absence of obstruction at the two arterial outlets of the heart.

95. In fine, the theory advocated by A. Leared appears more in accordance with the principles of physics, and better reconcileable with numerous facts of clinical experience (many of which will appear in the sequel) than any of its rivals. [629.] So that from the stand-point of existing knowledge, I should be inclined to accept the doctrine that the first sound of the heart is essentially the product of:

> Shock between the advancing blood from the ventricles and the momentarily fixed blood in the communicating arteries.

Then as a *constant* accessory of *noise* with systolic time must be admitted.

> Forcible shock of fluid against resisting tissue,—that of the blood against the orifices of the large vessels.

And as *occasional* accessory causes of *noise* with systolic time would rank,

> Impulsion of solid against solid—that of the heart's apex against the chest-wall.
>
> Abrupt friction of surfaces,—that of the two layers of the pericardium.

But, though an occasional cause of noise, systolic in time, impulsion of the heart against the chest-wall is not only not *the* cause of *the* first sound, as taught by Magendie, but that impulsion may, when soniferous, actually either partially cover or, on the principle of interfering vibrations, weaken the true first sound.

The influence of valvular vibration seems altogether questionable, and that of muscular contraction only hypothetically admissible as an accessory.

But, assuming that there are sufficient reasons for rejecting the long-accredited theories of valvular vibration and muscular contraction, and for accepting, as more likely to be true, another hypothesis in their room, it is consolatory to reflect that the uncertainty, such change of opinion shows must still hang over this part of the heart's *physiology*, does not carry with it corresponding uncertainty as to the *clinical signification* of various perverted states of the sound. A systolic murmur of certain attributes at the left apex, for example, will continue to signify reflux through the mitral orifice, no matter what changes of theory may be promulgated or accepted concerning the mechanism of the natural sound. But, on the other hand, it does carry with it of necessity more or less uncertainty as to the mechanism of those perversions. In this latter point of view the blood-concussion theory is the least unsatisfactory: it makes clearly intelligible, for instance, as shown by A. Leared, the change of a shock-sound into a murmur by diminution in the viscidity of the fluid.

96. *Mechanism of the second sound.*—The possibly soniferous phenomena, synchronous with the second sound, are: the diastole of the ventricles, and rush of blood into their cavities; the sudden recedence of the heart's apex from the chest walls; the abrupt fall of the auriculo-ventricular valves to the sides of the ventricles; the sudden tension of the sygmoid valves, and impulsive fall of the columns of blood against them during the arterial systole; the arterial systole itself.

(α) Now, powerful though the diastole of the ventricles be—sufficiently so even in the new-born infant to open out violently the hand closed around them*—it seems certain, as a matter of experiment on living animals, that the phenomenon is soundless. Negative evidence also abounds as to the non-production of noise by the flow of blood into the ventricles. Both occurrences may be looked on as practically non-soniferous in the normal state.

(β) If, in the natural condition, the sudden partial separation of the layers of pericardium, which had come together in systole, produce diastolic sound (and it would be difficult enough to prove a satisfactory negative), that sound is so slight as to be inappreciable to ordinary ears. In disease the case may be different [98].

(γ) I am not aware of any experiments tending to elucidate the part, if any, played by the fall of the auriculo-ventricular valves in generating sound of diastolic rhythm. There is certainly no evidence even tending to show that *the* second sound is in any measure thus produced, while *à priori* argument tells in the strongest manner against the possibility of such influence.

(δ) The absolute disappearance of the natural second sound at the aortic orifice, and its persistency (as can occasionally be proved, and hence as may always be inferred) at the pulmonary orifice in cases of *insufficiency of the aortic valves*, shows conclusively, as originally taught from clinical observation by Carswell,* that the sygmoid valves are essentially concerned in the generation of the true second sound.†

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* As noticed, for example, by Cruveilhier in his case of Ectopia cordis.—Gazette Médicale; Août, 1841.

This being conceded, the sygmoid valves may be held to generate the sound by their vibration at the moment of diastole, or they may be supposed to contribute to its formation through their influence in arresting the blood-current in its diastolic recoil towards the ventricles. The first of these notions is the more commonly received; but of the importance of the second, originally advocated, as far as I know, by Neil Arnott†, and subsequently, but independently, by A. Leared,‡ no doubt can be entertained.

That the fall of the column of blood on the sygmoid surfaces plays the sole part in the generation of the second sound is, perhaps, opposed by one principle in the mechanics of the heart commonly held by physiologists of the present day. It is taught that the valves are gradually opened out by the receding blood, which *then*, and not till then, in its mass falls with comparatively full, or at least tempered, force upon them. But Hammernjk, revising herein a forgotten opinion of Lower, maintains that the valves are first opened out, and thereupon receive abruptly and instantaneously the full weight of the recoiling blood column. If this were eventually proved to be the fact, the blood-concussion theory of the second sound would become unassailable.§

(ε) Whether the arterial systole itself contribute or not to the generation of *the* second sound must be admitted to be yet open to inquiry. But that the phenomenon is soniferous cannot reasonably be doubted; the occurrence of a double sound in the arteries, far distant from the heart, obviously settles the point.

(ζ) Although diastolic jogging action and quasi-impulse may occur without actual disease of the heart, yet there is no proof of such impulse being *per se* soniferous; the more so as, even in cases of strong morbid diastolic impulse, that impulse does not distinctly produce sound. Magendie's idea that the normal second sound is produced by the shock of the right ventricle against the sternum takes rank among the unquestionable errors of its day.

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* Archives Gén. de Médecine, 1831.

† Vide extract, p. 56.

‡ The Sounds caused by the Circulation of the Blood, 1861.

§ "If," observes A. Leared, in the experiment on the "leaden tube referred to by N. Arnott, the cock, instead of being turned suddenly, is so turned as to allow only a small quantity of water to pass through, a rustling continuous sound results. The change of the normal second heart-sound into a murmur from incompetency of the valves, is thus demonstrated."

97. Hence in the normal state the causes of the second sound would appear to be:

The recoil of the blood on the surface of the sygmoid valves;
The sudden tension of those valves.

And it seems infinitely probable that the former of these causes plays the essential, the latter a very subsidiary, part in the generation of the sound.

It will be perceived that, according to the theory of the two sounds now accepted as the least open to grave objection, *both* are produced, practically speaking, in the *same spot*,—at and a little above the level of the sygmoid valves. Why, then, are their points of maximum loudness at different spots? [86.] This certainly appears a difficulty; but it equally applies to the valvular theory; for the sygmoid and the auricular valves lie, for all acoustic purposes, on the same level [15]. It appears to be in some way an effect of conduction *within the chest;* for Cruveilhier, in his case of ectopia cordis, found both sounds were most intense at the base,—in so much that he suggests we should look to the region of the base for the causes of both sounds [*loc. cit.*].

98. *Morbid reinforcements and imitations of the second sound.*—It is altogether another question whether, in various states of disease, diastolic occurrences, naturally soundless, become sonorous, and so produce a second sound of their own,—or whether certain adventitious phenomena may in those diseased states produce an imitation more or less close of natural diastolic sound. Concerning this matter, Skoda argues, that as exceptional cases occur, in which the second sound is weak at the base, and loud at the apex, while there is no diastolic impulse against the chest wall to afford a possible explanation, the sound must originate in part in the ventricular region. He suggests that it may be produced either by, first, the stroke of the blood under special circumstances against the ventricular walls during the diastole of the heart: or, secondly, by separation of the heart's apex from the surface, against which it had been pressed during the systole; or, thirdly, by separation from the chest-wall of the portion of pericardium which had been driven against this during the systole.

As for the first suggestion: we have admitted that in the normal state, the blood enters the ventricles from the auricles, with a current so calm as to prevent audible sound from being thereby produced in the former cavities. But in cases of highly marked aortic reflux, blood falls with notable force into the left ventricle, and may conceivably generate sound. I have unquestionably heard, at the left apex, a distinct sound in more than one such case, while at the aortic base the ordinary murmur of reflux alone existed: cases of the kind would probably be more frequently met with, were it not for the loudness, and transmission to the apex, of the murmur evolved at the base.* I presume this intensified second sound cannot be the transmitted sound of the pulmonary valves, because I have found it stronger at the left than the right apex;† though it might certainly be argued that this greater intensity of diastolic sound at the left, might depend on greater intensity of diastolic murmur at the right, apex (murmur drowning sound); for, as I shall hereafter show, the rule in aortic reflux is, that the attendant murmur is better conducted down the sternum than along the left ventricular region.

The second and third suggestions are certainly plausible. And for this reason: if, while the ear is applied to a stethescope on the chest-surface, a neighbouring spot be percussed with the point of a finger, which is instantly withdrawn when the blow has been given, two sounds will be heard for each blow; the first strong, corresponding, of course, to the direct impulse; the second, very weak, to the removal of the finger from the point percussed. Now the recedence of the heart's point from the side is here imitated.‡

In all this the usual ingenuity of Skoda is apparent; but he appears to me to have lent his influence too much to establish the unwarranted conclusion, that because in certain states of disease the two last phenomena may produce noise of diastolic rhythm, *ergo*, they take part in generating the natural second sound.

An allied hypothesis, founded on the principle just explained, that the true second sound—essentially ventricular—is at least in part (it has even been affirmed, wholly), caused by abrupt diastolic separation of the surfaces of the auricular-ventricular valves that had been brought together in systole, counts few, if any adherents at the present day.

Some further facts, bearing on this part of the subject, will be noticed in the account of Reduplication of the heart-sounds [p. 73].

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* Flood, U. C. H., Males, vol. vii., p. 265.

† In a certain proportion of cases of mitral reflux with dilated hypertrophy, an intensified second sound may be found at the left apex;—its explanation will be sought hereafter.

‡ The result is still more satisfactory if the inner surface of the chest-wall be percussed in the dead subject.

II.—MODIFICATIONS OF THE HEART'S SOUNDS, OCCURRING INDEPENDENTLY OF CARDIAC DISEASE.

99. *Modifications occurring in the sounds at their site of production.*—(*a*) The description now given of the sounds of the heart applies especially to the male adult. The first sound is somewhat higher pitched and more ringing in females and in children than in males, and hence apparently more intense. In women, too, it is habitually more extensively audible than in men; but rather in all probability, from the acoustic conditions of the chest generally than as an intrinsic attribute of the sound itself.

(*b*) Variation of attitude has little, if any, effect on the second sound: but the first is commonly stronger in the erect than in the reclining, and in the prone than in the supine, postures: the reasons are obvious.

(*c*) The extent and direction of transmission of the natural sounds of the heart are modified by various changes in the conducting faculty of the structures and organs within, or on the confines of, the thorax; hence the obvious inference that, unless the absence of such changes has been established, we are not justified in inferring that a *plus* or *minus* extent of propagation depends on the state of the heart itself. Thus, as a ready illustration, enlargement upwards and inwards of the liver or spleen renders the sounds unduly audible in one or other hypochondrium; but alterations in the density of the lungs practically furnish the most striking and significant examples.

(*d*) When the heart is displaced by disease extraneous to itself, it might be supposed that the points of maximum force of its sounds would likewise be displaced: this is true, however, to a much greater extent of the first sound than of the second, and for the evident reason that the apex of the organ is more moveable than its base. In left pleuritic effusion, for example, when the maximum point of the first sound is carried to near the right nipple (say seven or eight inches out of its place), the maximum of the second scarcely swerves farther than to the right edge of the sternum, or one and a half inches from its post. Now it follows from this, that the line connecting the maxima points of the two sounds deviates, more or less extensively, from the almost vertical bearing it presents in health.

(*e*) The intensity of the heart's sounds is increased, and even the pitch of the first—heightened, by nervous excitement of all kinds: in the hysterical and epileptic paroxysm this is sometimes strikingly remarkable; emotion, whether of fear, anger, &c., has a similar effect, the sounds being in some instances audible even to the individual himself, and to by-standers at one or two feet distance from the chest. Diseases attended with marked debility weaken both sounds; but this effect is greatly more perceptible in the first than the second: the essential mechanism of the two sounds gives in each instance the clue to this difference.

(*f*) Certain conditions change the combined force and quality of the first sound by influencing some one of its elements without affecting the others. In continued fever the general weakness, impairing the ventricular power, lessens the force of the blood-shock at the arterial orifices, and gives the first sound a clicking character, akin to that of the normal second sound. Again, nervous excitement intensifies, and sometimes even gives a ringing, metallic quality to the systolic impulsive noise, produced by the projection of the organ against the side, so much so as to throw the real elements of the first sound into the shade. The second sound is comparatively little affected in either case.

100.—*Modifications arising in the sounds beyond their site of production.*—The changes, just noticed, occur at the spots of production of the sounds. Can the heart's sounds be increased in *force*, or changed either in *pitch* or in *quality*, subsequent to their generation, and through the influence of conditions beyond the heart?

(*a*) Both sounds may be materially louder in the site of a cavity under the left clavicle than at the mid-sternal base: and though rarer, when the cavity is seated at the right apex, such increase in amount of tone is even then not unknown. More than this, in a case of well-marked chronic consolidation of the apex of the right lung, *the second sound of the heart* (this organ, the arch of the aorta, and the pulmonary artery being, as far as ascertainable from signs and symptoms, perfectly healthy) *was decidedly louder under the right clavicle than at the mid-sternal base.* No such reinforcement existed at the sternal notch, nor under the left clavicle; the peculiarity was therefore not due to reinforcement in the arch of the aorta: and further, the mediastinum and apex of the left lung were, judging from their percussion-note, free from solid matter. Here, then, was a case where the second sound proved to be louder at about three inches distant from its point of production than at that point itself: the pitch of the sound in the two places was identically the same.[*] The mechanism of increased tone was here that distinguished as "unison-resonance."[†]

(*b*) Although it be theoretically conceivable that fall or rise in *pitch* of the heart's sounds shall occur independently of any other change, in the course of their conduction through varying media to the chest-surface, I know nothing of such modification as matter of experience, except under the circumstances referred to in the next paragraph.

(*c*) The condition of the hollow organs of the abdomen, especially the stomach, sometimes curiously modifies the *quality* of the heart-sounds. When that viscus is distended with gas to a certain amount, the sounds resound within it with a high-pitched metallic ring, and so loudly, sometimes, as to be perceptible to and seriously alarm, the patient.[‡] Large cavities in the adjoining lung (especially if their walls be hard, and the pleural sac distended with gas,) sometimes augment the heart's sounds by "unison-resonance," perhaps by echo, in a similar way: but in some instances the *pitch* of the sounds undergoes change.

101. The *number* of sounds corresponding to each revolution of the heart may be increased, but not diminished, independently of structural disease in itself, in the manners to be presently described [p. 73].

III.—MORBID STATES DEPENDENT ON HEART-DISEASE.

102. These are referrible to the heads of—I. Modified sounds; and, II. Adventitious sounds or murmurs.

I.—MODIFIED HEART-SOUNDS.

103. The intensity or loudness, and the extent of transmission of the sounds, when modified by heart-disease, stand in the direct ratio of each other.

(*a*) *Increase* in both respects is observed to its maximum in dilated hypertrophy, so long as the valves remain healthy. In this condition it may be held that muscular substance exists in sufficient excess to intensify the blood-shock causing the first sound, while the thinness of the walls, as compared with the size of the whole organ and the capacity of its cavities, favours resonance, and acts as a special source of loudness. Again, the forcible propulsion of the blood into the aorta leads to proportionally forcible reaction of the arterial coats, and unusually sharp recoil of the column of the blood on the sygmoid valves, hence a loud, full-toned second sound. In simple dilatation, the first sound, louder than in health, is sharper and more toned: the heart is brought more extensively than natural into contact with the chest walls, while, *possibly*, the form of the ventricles lends itself more effectively than in health to imperfect unison-resonance. Induration of the walls of the heart weakens the first sound, by lessening contractile power; it will further give peculiar intensity to the noise produced by impulsion of the apex against the side, if the other elements of that addition to the true first sound chance to be present.

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[*] In Allen, U. C. H., Males, vol. ix., p. 196, the same phenomenon was ascertained on the left side.

[†] Vid. Diseases of Lungs, 4th Edit., p. 144.

[‡] Warren, U. C. H., Males, vol. viii., p. 35; aneurism of arch of aorta.

Functional excitement of the organ also intensifies the sounds, whether such excitement be dependent on incipient inflammation of the organ itself or of its membranes, or on general pyrexial disturbance previous to the occurrence of debility. But excitement of purely *nervous* or *neurotic* dynamism, by increasing the force with which the blood is propelled, is the most powerful intensifier of the heart's sounds; it will even temporarily produce a loud first sound from a solidly hypertrophous left ventricle, and alone and unaided has much more frequently than any organic affection been known to render the sounds audible at a distance from the surface of the heart. I doubt if any structural change, unaided by nervous excitement, be competent to produce the latter phenomenon [99, *e*].

The quantity of blood circulating through the heart also modifies the intensity of the first sound. If the quantity be considerable, the struggle of the ventricle to force it on is considerable and prolonged; but the sound does not gain, indeed rather loses, in resonance and loudness. When the quantity is small, on the contrary, there is no such struggle; the ventricles contract abruptly and sonorously. It is under those circumstances that, as already mentioned [94, *y*], it has been suggested collision of the ventricular surfaces may intensify and modify the quality of the systolic sound.

(*b*) The sounds are *weakened*, where the muscular structure of the heart is encroached upon by morbid infiltrations, or deteriorated by its own disease. Softening, obesity, fatty metamorphosis, cancerous infiltration, atrophy with fibroid infiltration, all impair its force. In dilatation with attenuation, the first sound, though sharp-toned, is feeble—probably from conjoined change in the sarcous structure.

In simple, and still more in concentric hypertrophy, of the left ventricle, the first sound is weakened at the apex in, it may almost be said, the direct ratio of the increase in mass of the muscular substance and decrease of the cavity. In extreme cases, the first sound may be completely deficient over the left ventricle and perceptible only at the base, or towards the ensiform cartilage. The compact packing of the muscular structure may be supposed to interfere with freedom of contraction, and the diminished size of the cavity obviously must impede the play of its parietes. Even valvular action is diminished in scope, and while the mitral valve is of less superficial extent than fairly proportional to the increased thickness of the ventricle, it is often thicker than natural. The entire mass of the ventricle, too, may not contract simultaneously. But whatever be the theory adopted of the mechanism of the first sound, its diminished tone in pure hypertrophy remains difficult of comprehension.

Accumulations in the pericardium of fluid, of air, or of air and fluid combined, weaken (as well as otherwise alter) the sounds by removing their seats of production from the surface. I do not know clinically the effect of solid formations in the pericardium; they may be conceived to weaken the sounds in one way, and intensify them in another,—severally in regard of production and of conduction.

Local want of muscular and nervous tone, associated or not with systemic feebleness, weakens the first sound.

In some rare cases that fall under our notice, *while the first sound has proved so feeble as to be scarcely audible, the second has been full-toned, strong, and audible over the entire chest.* The notion of a hypertrophous condition of the pulmonary artery suggests itself as a possible explanation; but I have no certainty of knowledge on the matter. In the cases referred to, none of the commonly admitted affections of the heart or great vessels existed.

(*c*) The *duration* of the *first sound* falls below the natural standard in dilatation without hypertrophy, or with attenuation, and under all circumstances which shorten the period of muscular action. The sound is lengthened more or less in hypertrophy, with moderate dilatation; and if, in addition, the mass of circulating blood be large, or if the aortic orifice be at all obstructed, the sound may be sufficiently prolonged to fill nearly two-thirds the period of each revolution of the heart.

The *second sound* is shortened by a thin paper-like state of the sygmoid valves and by thinness of the blood; its pitch rises under the same influences. The sound is lengthened, on the contrary, by thickening of the valves, and a loose inelastic condition of their texture. Probably, too, thickening of the non-striated muscular portion of the arterial walls will lengthen their own systole, and hence, probably, the diastolic heart-sound. Another curious cause of prolongation of this sound will be by-and-by described.

(*d*) The points of maximum intensity of the heart's sounds are more liable to *displacement* by extrinsic than by intrinsic causes; the latter, however, in some forms influence them. Enlargements of the heart generally, whatever be their nature, *lower* the maximum point of the first sound. Simple, and *à fortiori* concentric, hypertrophies influence its position much less, in proportion to their mass, than the eccentric variety of the disease. Pericardial effusion, when it raises the apex, *raises* the point of maximum intensity of the first sound; and in some rare instances I have known this carried backwards, the sound being more distinct in the left vertebral groove than in the ordinary apex-region. Hypertrophy of the right ventricle lowers its own first sound; of the left, carries its own immediately to the left, as well as depresses it. In all cases of depression of the first sound, the second is similarly affected, but to a less degree. Marked eccentric hypertrophy of the auricles would also probably lower the first more than the second sound; but I do not know this from experience.

(*e*) The *distance* of the heart's sounds from the ear of the auscultator is very manifestly *increased* in cases of fluid and gaseous accumulation in the pericardium. There is, however, an occasional source of fallacy here; the sounds are commonly listened to in the natural situation of the apex, and not in the neighbourhood of the nipple or fourth rib, whither that apex has been carried by the disease. Agglutination of the pericardial surfaces, on the contrary, brings the sounds *nearer* than natural to the ear; if not precisely at their maximum points, beyond these. Enlargement of the heart, sufficient to push away the left lung, will have a similar effect. The first is more affected by these changes than the second sound.

(*f*) The *quality*, and with this the *pitch*, of the heart's sounds is subject to serious modifications.

The *first* sound becomes dull, muffled, toneless, and in some cases almost null, at the apex, where dense hypertrophy is conjoined with a thickened inelastic condition of the auricular valves. On the other hand, where the ventricular walls are thin and the valves natural, the first sound becomes more or less clear, flapping, or clicking, with raised pitch; if those walls be in a state of eccentric hypertrophy, and the valves somewhat thickened, the sound assumes a clanging sharacter. At least, these statements are in accordance with the majority of results; but exceptions, explicable, perhaps, sometimes by the state of the heart's texture, —in others inexplicable,—pretty frequently occur. It would be difficult to describe or explain, for instance, the varieties of quality found in the first sound of soft, flabby, fatty hearts, with healthy, or nearly healthy, valves. Elevation of pitch is associated, as a rule, either with thinness of the muscular walls, or with a watery state of the blood.

The first sound sometimes possesses a peculiar full-toned quality, without the least sharpness, while it is strongly accentuated at the commencement, and commonly prolonged: the nearest articulate symbol of the two sounds, under these circumstances, appears to be *b'oom-tup*, pronounced with strong emphasis on the *b*. I have observed this peculiarity in cases of eccentric hypertrophy of the left ventricle,—but without ascertaining the special condition on which it depends.

The quality of the systolic sound at the apex seems sometimes sharply knocking; but with care this knocking quality is separable by the ear from the true heart-sound, and obviously depends on the impulse of the apex against the side,—but not necessarily against the inferior border of the fifth rib, as imagined by Hope. Knocking impulsive sound cannot be called an essential or even habitual attendant on any particular disease of the heart; neurotic palpitation, especially if the edge of the lung be by some disease of its own carried unduly towards the left, will readily produce it in a structurally healthy organ. Thin-walled resonant chests supply it with greater ease than others; and morbid induration of the heart's apex, or calcification of the pericardium, will aid in intensifying it. The heaving and steadily pushing character of the impulse in simple hypertrophy prevents its occurrence in that disease: eccentric hypertrophy is the form of enlargement it most frequently accompanies.

The *first* sound is sometimes slightly rough, and approaching in quality to a murmur at the apex; it is, in fact, *murmur-like*, without being actually converted into a murmur.[*] This may be (α) a persistent condition, observable week after week, while the patient remains under treatment; or (β) a temporary state, constantly noticeable for a few days, and then disappearing; or (γ) a mere transient phenomenon, occurring with some, absent from other, beats of the heart. In the first case (α), it has appeared to me referrible to an incipient or slight amount of some one of the organic conditions which, carried further, produce a perfect systolic murmur. Can it ever be produced by a buzzing murmur-like quality in muscular sound, produced by slow, drawling contraction of the fibres, or some special alteration of their texture? The second case (β) is exemplified by some *excessively rare* instances of acute rheumatism, where the systolic sound at the left apex, roughened and murmur-like for a few days at the outset, then loses this quality, either permanently, or to resume it again at a later period in a more decided form.[†] Passing vascular roughness of the mitral, or even ventricular, endocardium, and imperfect closure of that valve from the influence of irritation, suggest themselves as possible causes of the phenomenon; but anatomical evidence is *ex naturâ rerum* wanting on the point. In the third case (γ), the peculiarity is caused either by coincidence of the respiratory sound with that of the heart,[‡] or by rubbing of the apex against the pericardium,[§] or by movement of air in the adjoining lung-substance produced by the cardiac impulse, or (the most important cause, because the most likely to lead to error) by a tendency to reduplication of the first sound.

The *second* sound at the base is rendered dull, and, so to speak, *thud-like*, by fibro-fatty thickening, without insufficiency, of the sygmoid valves. Diminished elasticity of the arterial walls has a similar effect.

Like the first, the *second* sound may be murmur-like, temporarily or permanently. Very trifling insufficiency will probably thus modify its quality; whether marked reticulation of the valves may suffice for the purpose, will hereafter be discussed. The most common cause of murmur-like quality in this sound is a tendency to reduplication.

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[*] In the case already referred to [93, δ], when the contraction of the abdominal muscles occurred in a slow vermicular manner, the sound was distinctly of murmur-like quality.

[†] James Hayes, U. C. H., Oct., 1850.

[‡] It is sometimes impossible to satisfy oneself on the influence of this coincidence, unless by causing the patient to suspend his breathing.

[§] The more superficial character and the influence of change of posture will generally distinguish this variety from a true murmur.

(*g*) The natural *accentuation* of the sounds, as shown in a previous page, is liable to numerous perversions; but as the accent falls on whichever sound is intensified, and mainly at the spot of intensification, repetition may be saved by referring the reader to the paragraphs on augmented intensity. When the heart is weak and flabby, either from organic change, or from want of tone, there may be a total deficiency of accent on either sound at the apex; the sounds resemble those of a vibrating pendulum.

Or the normal spot of accentuation may be displaced. Thus the second sound is, in certain rare cases of mitral regurgitation, so intensified that the accent falls on it, even when auscultated at the heart's *apex*. And, again, in fatty metamorphosis, the accent may, and commonly does, distinctly fall on the diastolic sound *at the apex*.

(*h*) Like the sounds, the *silences* vary in disease in relative duration. The first, or post-systolic, silence is lengthened by deficient elasticity of the arterial walls, whereby the recoil of the blood on the valves is sluggishly effected; so, too, whenever the first sound is disproportionately shortened, the first silence is lengthened. The first silence is normally so short, that it is difficult to appreciate its decrease.

The second, or post-diastolic, silence is lengthened in cases of advanced constriction of the mitral orifice; the process of filling the ventricle is laborious and slow, and hence the systole lags, as it were, behind its time. When the circulation is greatly slackened, the second silence is generally disproportionately prolonged.

(*i*) The *relationship of the sounds of the heart to the pulse* varies in disease.

In the normal state, the first sound is apparently synchronous with the diastole of the arch of the aorta, the pulmonary, carotid, and subclavian arteries; thenceforth, the further the vessel from the heart, the more distinct is the interval between the systolic sound of that organ and the arterial diastole. It is difficult to determine the possible length of interval consistent with health: but it may be affirmed, that if the diastole of the most distant vessels, as the posterior tibial and dorsal artery of the foot, is so much retarded as to become synchronous with the second sound, the state is morbid. This retardation, which was first noticed by Henderson as an attendant on insufficiency of the aortic valves, may with care be detected in many, but unquestionably not in all, cases of that disease. Possibly where no morbid retardation can be discovered, the failure may depend, not on its absence, but on its being carried to such extremes, that the arterial pulse produced by one cardiac systole is *quasi*-synchronous, or actually synchronous, with the next. The only fact, however, I know of, supporting this idea, is, that it is in *extreme* cases of aortic regurgitation the pulse seems most disposed to stand in normal relationship of time to the heart's systole. The same sign exists in attenuated dilatation of the left ventricle also.

Again, in health, the frequency of the pulse and the length of the systolic sound vary inversely as each other: a frequent pulse is the index of a short first sound, and *vice versâ*. The same relationship holds good in some morbid states; for example, in anæmia and in the re-action after hæmorrhage. It is, on the other hand, occasionally perverted; the pulse may be infrequent, and the systolic sound short: in fatty metamorphosis, and in simple flabby softening of the heart, this perversion may commonly be noticed.

The sounds of the heart are sometimes suspended for the precise length of time occupied by an ordinary revolution of the organ: they are said then to *intermit*. Not uncommonly such intermission recurs with considerable regularity; that is, after a fixed number of regular beats. Sometimes the systolic sound seems to *anticipate*, sometimes, on the contrary, to *hesitate* at, the proper moment of its occurrence,—changes of rhythm closely connected with shortening or prolongation of the second silence. Sometimes a series of feeble and rapidly succeeding sounds follows others comparatively loud, slow, and deliberate: and there may be a certain uniformity in the number of each kind, and in the periods of their recurrence. Or the *irregularity* of the sounds may be complete, both in intensity and in rhythm, no two revolutions corresponding to each other in either character: there ceases to be any semblance of order in disorder. This excessive perversion exists in certain rather exceptional cases of highly marked mitral regurgitation, in extreme softening, fatty infiltration, acute destruction of a portion of a valve or of chordæ tendinæ, rupture of these structures, formation of fibrinous coagula within the heart, and in a small proportion of cases of pericarditic effusion.

The natural correspondence in the number and time of cardiac systoles and arterial pulsations is habitually maintained, even when the rhythm of the heart's contractions is thus variously altered. If the left ventricle intermits, or anticipates, or hesitates, or becomes wholly irregular in its contractions, a precisely similar change occurs in the arterial pulses: the impulses of the connected tubes are the counterparts of those of the central organ. But, on the other hand, there may be a failure of this correspondence, not only when the heart's contractions are thus abnormal in rhythm, but even when they are in this respect normal. Thus two revolutions of the heart may correspond to a single radial pulse, the cardiac action and the pulse being perfectly regular in force and rhythm; or the pulse may be perfectly regular, and the heart's successive systoles somewhat unequal in force and duration, as in a case formerly recorded,* where eighty-eight systolic contractions produced forty-four radial pulsations. Here the rhythm of each pair of beats of the heart might be represented thus:—Systole = 5, diastole = 3; systole = 9, diastole = 4. It was the first of the two systoles that failed to affect the pulse at the wrist; and as there was no evidence of aortic or mitral disease, but merely of flabby enlargement, that systole may have been dynamically incapable of influencing the distant vessels: the state was of temporary duration. I have observed a similar condition, persistent, but of less regular type, in cases of extreme contraction of the mitral orifice: under these circumstances, doubtless the systole occasionally takes place, before the ventricle is supplied with blood to propel.

Again, in certain cases of utter irregularity of the sounds there may be no traceable accordance between them and the force or rhythm of the pulse. This is, perhaps, best observable where the irregularity comes on suddenly from rupture of a valve, or accumulation of coagula in the cavities: but is occasionally met with in all the diseased states productive of rhythmic irregularity. Generally speaking, in instances of this form of discordance, the heart's action and arterial pulse are frequent; but a very remarkable example of utter want of synchronism between cardiac and arterial impulses has recently fallen under my notice, where the pulse commonly oscillated from 32 to 38 per minute, and occasionally fell to from 26 to 30. This case, seen only in life, appeared to be one of specific motor neurosis combined with deterioration of fibre probably fatty in nature.

(*k*) The *number of sounds* attending each beat of the heart may vary, the arterial pulse holding its natural relationship to the systole.

(*l*) *Number of sounds diminished.*—A *single* sound only may be heard, and this may be the first or the second; whichever sound be deficient in any particular spot, it may, or may not, be audible at some other part of the cardiac region. The first sound may be quasi-deficient at the left apex, when the conditions, already described as weakening it, are carried to extremes; but it will then be found at the right apex, and at the base. So, again, the second sound may be quasi-deficient at the base from excessive feebleness, or from being covered by a prolonged systolic sound, or systolic murmur: but in the first case, excitement of the heart, increasing the energy of its contractions, will invigorate the sound; and in the second case, the sound will be heard at the right apex. Absolute deficiency of either sound, or of a murmur taking its place, has never fallen under my observation; in other words, *neither systole nor diastole has ever been, in my experience, absolutely noiseless over the entire cardiac region.*

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* Clinical Lectures, Lancet, 1849, vol. i., p. 443.

(2) *Number of sounds increased, so-called reduplication.*—The systolic or diastolic sound, or both of them, instead of being single may be double; the affected sound is then said to be cleft, doubled, or reduplicated. The phenomenon is far from uncommon in some of its forms.

Such slight accentuation, as exists, falls usually on the first of the three or four sounds; the pitch of the two divisions of the doubled sound is sometimes distinctly different. Reduplication existing at one spot may be audible elsewhere, or limited to that spot. The forms of reduplication, which have fallen under my notice, are the following:—

1. *First sound.*—Double at the left apex (*ŭbbŭp-dŭp*), at the same time single or double at the right apex, and single,[*] or, more commonly, double, at the base.
2. *First sound.*—Double at the right apex (*ŭppŭp-tŭp*), single at the left apex and at the base.
3. *First sound.*—Perfectly doubled at the base, imperfectly so at the tricuspid apex, single at the mitral apex.[†]
4. *First sound.*—Double at the base, or at the apex, while the second sound is conversely double at the apex or the base.
5. *First sound.*—Double at the left apex, single at the base; two days later the doubling inaudible at the base and at the left apex, but distinct at the right apex, or upper edge of the ensiform cartilage.[‡]
6. *Second sound.*—Double at the base (the most common of the series), very generally single at the apex; the reduplication audible or inaudible at the pulmonary and aortic cartilages both, or audible at either singly.
7. *Second sound.*—Constantly double at the tricuspid apex, and single at the base and left apex:[§] or, while constantly double at the tricuspid apex, occasionally so at the base.[‖]

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[*] Bowry, U. C. H., Males, vol. vii., p. 259.
[†] Branchley, U. C. H., Females, vol. ix., p. 94.
[‡] Lenton, U. C. H., Males, vol. x., p. 86.
[§] J. Hayes, U. C. H., Males. Oct. 17, 1850.
[‖] W. Hodson, U. C. H., Males, vol. ix., pp. 65, 66.

8. *Second sound.*—Constantly double, beat after beat, at the mitral apex; reduplication less distinct at the tricuspid apex; imperfectly or not at all reduplicate at base, and always attended with faint murmur (the difference in the amount of reduplication at the base and mitral apex is very striking); slightly reduplicate at pulmonary, not at aortic cartilage.*

9. *First and second sounds.*—Both reduplicate (*ŭppŭp-tŭrrŭp*). This is very rare, is more frequent at the left apex than elsewhere, and has never appeared to me audible at all parts of the heart's area.

There are, besides, occasionally observable curious modifications of the phenomenon of which the following are perhaps the most remarkable. Reduplication may change from one sound to another.† Doubling of the second sound at the base may be arrested by deep inspiration: or the second sound at the pulmonary cartilage, single in expiration, may be most distinctly split into two at the end of inspiration.‡ Sometimes, again, a tendency to reduplication exists without the sound being actually double; this state of things prolongs, and may give a murmur-like quality to, the affected sound [103, *f*].

The essential *cause* of these various reduplications seems to be a want of synchronism between the actions of the two sides of the heart. If the facility with which the two ventricles fill with blood be unequal, they will probably differ somewhat in their time of contraction; certain conditions of the auriculo-ventricular valves may bring their closure on either side, as it were, behind time: in either of these contingencies the production of a double systolic sound at the apex becomes readily intelligible. But the fact that such reduplication may be audible at one apex only, and perfectly inaudible at the base, is far from being easily explicable; it cannot, except on the merest hypothesis, be referred to double spasmodic contraction of either ventricle, and in point of fact did such contraction occur it would be attended in great probability with double systolic impulse, and this I have failed to detect in cases of the sort. Again, as originally observed by Charcelay,§ the contraction of the auricles, when highly hypertrophous, may become sonorous, and so double the first sound; but it is scarcely necessary to add, that such mechanism must be of singular rarity. Nor does it appear probable that the characters of the double sound could, under the circumstances, resemble those of ordinary reduplication: thus in a case of hypertrophy of the left auricle, a loud præsystolic sound of maximum force at the third left cartilage differed absolutely from a reduplicate sound by its special *knocking* character.*

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* Couch, U. C. H., Females, vol. vii., p. 343. The above true, as far as the words "at base," of Dunn, U. C. H., Females, vol. xvi., p. 296.

† Branchley, U. C. H., Females, vol. ix., p. 146. "At base systolic sound doubled, when first listened to; within two minutes the second reduplicated: no doubling of either sound at left apex during examination."

‡ S. Roberts, U. C. H., Females, vol. v., p. 220.

§ Archives générales de Médecine, 1338, p. 393.

Similarly an imitative reduplication of the first sound may sometimes be produced by the knock of the apex against the side; but the least practised ear will readily distinguish this from true *intra-cardiac* reduplication.

Non-synchronous recoil of the arterial blood-columns on the sygmoid valves, again, easily explains the reduplication of the second sound at the base; and may itself be referred to unequal elasticity in the coats of the aorta and pulmonary artery, unyielding stiffness of either set of valves, a material obstruction in the way of their closure, or any state of either ventricle rendering it slightly tardy in the propulsion of its blood into the artery beyond. The fact that inspiration, especially if full,† will sometimes cause doubling of the second sound, which is inaudible in ordinary breathing, may be explained by the unduly abrupt rush of blood into the pulmonary artery, whereby the necessity for closure of its valves, to meet the recoiling fluid, is felt a little earlier than usual. But the same difficulty, as in the case of reduplicate systolic sound, arises in accounting for the limitation of the phenomenon to certain points of the cardiac region.

In regard of *Diagnostic Signification* it must be confessed, these reduplications are almost valueless in the present state of knowledge. And for the following reasons:—reduplication is never, as far as I have observed, permanent and invariable; it occurs most commonly in hearts either healthy or temporarily disordered in function only: less commonly in cases of slight organic affection; and with least frequency when serious valvular disease exists;‡ it is not connected, as a rule, with any particular form of disease, either of the heart itself, or of the system, rheumatic or other; it comes and goes in the course of a few beats of the heart; sometimes disappears on change of posture, and may be affected, nay induced, by the act of respiration.

At the present hour the main interest of reduplications arises out of their bearing on the theory of the heart's sounds,—a fact of which the following illustrations afford sufficient evidence.

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* Fred. Smith, U. C. H., Males, vol. v., pp. 297-8, Nov., 1850.

† Shillingford, U. C. H., Females, vol. xiii., p. 110.

‡ It is fair to observe, however, that the existence of murmurs in such cases may be the real cause of reduplication not occurring, or not being heard.

(1.) The second sound may be continuously doubled at the base, and perfectly pure and single at the apex. How is this explicable on the simple sygmoid blood-recoil theory of the second sound? Does a double sound become single by conduction over so short a space?*

(2.) The first sound may be single at the left apex and at the base, while it is distinctly reduplicate at the right apex. Can it be that under these circumstances the true first sound at the tricuspid side is *quasi*-doubled by some accidental element of sonorousness (say, impulse of the blood against the orifices of the great vessels [94, η]); or, as suggested by A. Flint, is there simple failure of conduction to the left of the naturally feebler triscupid† segment of the first sound?

(3.) The second sound may be double at the base, and single at the aortic, double at the pulmonary, cartilage, or *vice versâ*. This cannot arise from want of synchronism of recoil on the two sets of valves, but on one or other of the three divisions of one set.

(4.) The second sound may be single at the base and double at the left apex; now, according to the pure sygmoid-recoil theory, that recoil is the sole source of the second sound: how come the two sets to divide their compound sound at the apex? Splitting into two, as a result of conduction from the base to the apex by ventricles of different conducting powers, cannot be admitted; for the reduplication may be present at the left apex only, absent at the right. This is the strongest fact, I know of, in favour of the second sound being in some cases partly of ventricular origin [97].

Possibly some of the difficulties in explaining these reduplications might be removed by giving due weight to the facts, concerning the time-conduction of murmurs (change of synchronism at base and apex), referred to a little further on [106, *j*].

II.—ADVENTITIOUS SOUNDS, OR MURMURS.

104. Sounds of adventitious origin and properties, produced either within or on the surface of the heart, may be conventionally termed *murmurs*. In applying the term to any sound heard in the cardiac region, we imply that the mechanism, statical and dynamic, singly or combined, as likewise the intrinsic properties of that sound, differs from those of the true heart-sounds.

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* A. Flint (on Dis. of Heart, p. 279, 1859) thinks this fact explicable by the non-conduction, so far as the apex, of the naturally weaker pulmonary portion of the reduplicate sound.

† In one case (James Hayes, U. C. H., Oct. 17, 1850) where this form of reduplication was well marked, pericardial adhesions had recently formed.

105. In reference to their seat of production, cardiac murmurs are divided into endocardial and peri- or exo-cardial.

A.—ENDOCARDIAL MURMURS.

106. (*a*) The *special character* of all endocardial murmurs is, more or less, blowing. Their quality varies extremely, and they may be called simply blowing, grating, filing, rasping, sawing, whistling, or cooing, according to their greater or less similarity to these noises. The simple blowing murmur, though itself presenting different degrees of harshness, is always soft in comparison with the filing, grating, rasping, and sawing varieties. In some cases murmurs are real tones capable of musical notation.

(*b*) Endocardial murmurs may become audible to the individual himself (*auto-audible*); they may in some instances be heard by the observer at short distances from the chest.*

(*c*) They are essentially *intermittent;* and, no matter what its duration be in relation to the heart's contractions, a single murmur is never sustained continuously, either in a uniform or remittent manner, through a series of beats.

(*d*) In point of absolute *duration*, they vary from a scarcely appreciable moment to two or three seconds: the latter amount of prolongation can only occur where the physical conditions are at once peculiarly favourable for sustainment of sound, and the action of the heart extremely slow.

(*e*) The *pitch* varies by several notes, the lowest being, perhaps, represented by the whispered word *who*, the highest by *ss:* intermediate notes may be represented by whispering the word *awe* by inspiration, and the letter *r*, with various degrees of closeness of the isthmus of the throat: these are the suggestions of Hope.

(*f*) In point of apparent *distance from the surface*, they vary also: so deep, in some cases, as obviously to be weakened by distance; in others they seem to originate directly under the skin.

(*g*) The spots of *maximum force* of individual murmurs, the position of the heart being normal, are four,—a few lines above the left apex; just above the ensiform cartilage; at mid-sternum, on the level of the third interspace; and at the junction of the third left cartilage with the sternum. But these points of maximum force are liable to change, both from various malpositions of the heart, and from alteration in the conducting qualities of the materials around it; the laws of transmission are then the same as in the case of the healthy sounds: a special cause, the direction of the current of blood, will be considered hereafter.

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* When the last edition of this book appeared, I had only once to my recollection met with this; namely, in a cyanotic child about six years old, in whom a systolic rough blowing murmur could be heard at a distance of an inch or a little more from the surface. I have since twice observed the peculiarity in the adult—in cases where loud murmur, diffused almost through the entire arterial system, suddenly came on under peculiar circumstances.

(*h*) An endocardial murmur once developed is habitually *persistent*, so long as it lasts; that is, it does not come and go, but attends every beat of the heart: however, weak systoles may fail to produce a murmur, which is well marked with strong ones; tendency to syncope, general debility, and collapse, and the approach of death, may prevent its production; dynamic murmurs are sometimes intermittent; and sometimes (for instance, when the cause of the murmur is pressure on an arterial orifice by a tumor) certain postures may annul a murmur completely.

(*i*) The *influence of lapse of time* on murmurs varies. It may intensify, lessen, or remove them. Besides, they may or may not attain a higher type of harshness the longer they exist: again, a fall in pitch sometimes takes place as time rolls on. A musical murmur may be converted into a harsh toneless noise; or a musical murmur may totally and suddenly disappear, in infinite probability from the dislodgment of some attached vibratile fragment. Under these circumstances the cessation of the murmur may or may not be followed by evidences of distal embolic obstruction: both contingencies have fallen under my notice.

(*j*) The *rhythm* of murmurs may be considered in respect of the heart's contractions and of the heart's sounds. In respect of the former, they are said to be systolic and diastolic; in respect of the latter, synchronous with the first or second sounds. But they are not necessarily synchronous with either systole or diastole. They may be pre-systolic, systolic, or post-systolic; pre-diastolic, diastolic, or post-diastolic: they may occupy a portion only of either time; or fill this and the succeeding silence, and encroach upon the succeeding sound,—as is common with systolic, rare with diastolic, murmurs. This arrangement is doubtless open to the charge of hyper-division; but as it positively has its foundation in correct clinical observation, it ought to be kept in view, although for ordinary purposes the simpler one is quite sufficient: there can be little doubt that many of the alleged failures of the rules for valvular diagnosis are traceable to inattention to these subdivisions of systolic and diastolic time.

The synchronism of a murmur, the student must remember, may or may not be easily defined: when doubt exists, the finger should be applied to the fifth interspace over the apex-beat, or to the nearest available artery,—the arch of the aorta at the sternal notch in some persons, or the carotid. If the murmur in question be precisely synchronous with the ventricular or arterial beat, its rhythm is systolic; if not so synchronous, the degree of non-synchronism will guide to the determination of its time. Now, as a rule, basic murmurs are easily timed: systolic, diastolic, or slightly anticipating, or lagging behind, systole or diastole. But this facility is far from being equally shared by apex-murmurs. Whether an apex murmur is purely systolic, pre-systolic, diastolic, or tailed on to the diastole, is sometimes a problem so perplexing that it may be solved differently by two or more persons of equal and of tried experience. The more rapid the action of the heart, the greater, of course, the difficulty.* In cases of very tumultuous and rapid action, digitalis may be used to slacken the beat.

A murmur may fail to present the precisely same synchronism at different spots of the chest-wall, even within the limits of the deep-seated cardiac region [11]. Thus, the rhythm of a reflux aortic murmur, precisely diastolic at the base, may cease to be so at the ensiform cartilage,—as also, if audible there, at the left apex. At these spots it synchronises more with the long pause, and approaches the first sound, or inclines to trench on systolic time.

(*k*) Endocardial murmurs are further distinguishable in respect of their *relationship to the blood-current;* whether systolic or diastolic, they may occur in the direction of the current of blood, or against it,—synchronously with onward movement, or with recoil of the fluid. Thus the systole of the left ventricle may produce a murmur at the aortic orifice with the current, or at the mitral orifice against it: hence a division of murmurs into direct and indirect,—which are also severally called, from their common causes, murmurs from constriction and from insufficiency of valves, or from obstruction of, or regurgitation through, orifices.

(*l*) The *effect of endocardial murmurs on the sounds* varies. A murmur may simply render a synchronous heart-sound obscure, at its commencement, at its close, or throughout its entire duration; or it may completely mask this by its intensity, and even similarly affect the succeeding sound; or it may prevent the natural heart-sound from being formed. Thus at the left apex a systolic murmur may completely drown a systolic sound, which is readily audible at the right apex and at the base. The systolic sound is not masked there, but actually deficient when: first, both auricular orifices are not in a condition to allow of the production of natural sound, but of murmur alone,—secondly, when the arterial, or basic, portion of the first sound is feeble, or itself converted into a murmur,—and, thirdly, when the muscular texture is seriously weakened by malnutrition or motor neurosis.

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* On this matter W. Stokes has some very strong observations, well worthy of consideration, even by those who may not share his scepticism (Dis. of Heart, p. 180, 1854).

When, as is very common, both murmur and sound are distinguishable coetaneously, the state may be called one of pseudo-reduplication. But true reduplication of a murmur is so excessively rare, that I remember to have met with but two examples of the fact: one, basic and diastolic, must have been in the aorta alone, for the pulmonary artery was unaffected; of the other, systolic at the left apex, I had no opportunity of examining the mechanism by dissection;—it might have been mitral and tricuspid together, or it might have depended on co-existing deep-seated venous hum,—in which case, of course, it was a false reduplication only. I once heard a post-diastolic murmur and sound in a rheumatic woman, in whom the doubling appeared to arise in consequence of the set of sygmoid valves, which gave the murmur, acting after the set which gave the sound.

(*m*) Some murmurs are unaffected, others affected more or less seriously, by the *posture* of the patient. A systolic murmur limited to the left apex, and highly marked in the sitting or erect, may totally disappear in the supine, posture, instantly re-appearing when the patient again sits up. Basic systolic anæmic murmur will often cease in the erect, to return in the lying, posture. Systolic murmur, strongest at the left apex, and powerfully marked in recumbency, may disappear in the erect posture, remain inaudible for several beats after the patient has again laid down, and very gradually recover its previous intensity;* or the exact converse may occur.

Is this changeableness of a murmur proof of its inorganic nature,—that is, does it prove that the murmur is, as matter of necessity, either of hæmic or of dynamic mechanism? My experience positively supports the negative. And if this experience has not deceived me, it would follow that certain statical defects at an orifice may dynamically be nullified by some unknown alteration in the relationship of the parts concerned, brought about by change from vertical to horizontal position of the heart, or *vice versâ*.

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* Constable, U. C. H., Females, vol. ix., p. 96. Here the patient was spanæmic, the aortic valves slightly diseased, the mitral not obviously unsound,—but whether regurgitation might or might not have occurred during life is matter of doubt.

(*n*) *Mode of generation.*—Where endocardial murmurs have existed, structural changes, dynamic perversions, or morbid conditions of the blood are found to explain them, hence their division as follows :—

- (A) Organic or structural.
- (B) Inorganic or non-structural
  - Hæmic
    - Of changed composition.
    - Of coagulation.
  - Dynamic.

(A) *Organic Endocardial murmurs* are essentially connected with such alterations of the orifices or of the valves, as, while they lead to constriction or imperfect closure of the orifices, cause unnatural friction between the blood and surfaces.

The chief of these alterations *may be arranged as follows :*—Simple *constriction*, or constriction with thickening, hardness, rigidity, calcification, warty or other excrescences from, or even simple inflammatory loss of polish and roughness of, the valves.

Simple *insufficiency* of the valves to close a widened orifice, themselves not having grown *pari passu* with the widening; or such insufficiency depending on the various diseases of the valves just enumerated; or depending on shortening and thickening of the chordæ tendineæ; or on atrophy or contraction of the columnæ carneæ; or on puckering of valves; or on adhesion of the divisions of a valve *inter se*, or to the adjacent surface.

Excrescences or other forms of thickening or calcification of the valves, unattended with positive insufficiency or constriction.

Attachment of a tendinous cord in a wrong situation, whereby it is thrown directly across the blood-current.

Unnatural communication between the different compartments of the heart, or between these and the arteries or some adventitious cavity.

Besides, without distinct structural alteration of the orifices or valves, *mere vascular roughness of the ventricular endocardium* probably suffices to affect the purity of the sounds, when the current bears, especially towards the arterial orifices, on such a roughened spot.

Coagula among the columnæ, or a *polypoid body hanging from the neighbourhood of the valves*, will have a similar effect.

I believe I have seen a case or two in which a puckered state of the heart's wall close to the mitral valve, through the influence of contracting pericardial exudation-matter, has so disturbed the mechanical as well as the vital play of the valve as to cause reflux-murmur. But such mechanism must be very rare.

Physically speaking, then, there appear in the main pure *constrictions* of natural orifices, pure *widenings* of orifices, and pure *roughness* of surfaces, to explain the ordinary mechanism of organic cardiac murmurs. Now pure constriction and pure roughness are positively capable of producing murmur: this is matter of experimental demonstration; by pressing with the stethoscope on an artery, we convert the dull fillip-like natural sound of the vessel into a whiffing murmur. But how can pure widening of an orifice produce murmur? In the case of the tricuspid and mitral orifices, the *regurgitation* from such a physical cause may intelligibly produce murmur by the collision of direct and indirect blood-currents coming from and going back into the auricle: in the case of a *direct* current through a simply widened aortic orifice, the rippling of the stream, produced by the change of calibre, seems, from the experiments of Corrigan, sufficient, if that current be strong, to produce murmur.

Mere alteration in the direction of the current, of a kind to throw the blood obliquely against, instead of carrying it directly through, an orifice, will theoretically generate murmur. Probably this divergence of the stream plays a part in many valvular murmurs produced by the onward blood-current; and as dilatation of the ventricles renders these more spherical and less convergent to their arterial outlets, it has been urged by Blakiston and others, that such state must produce murmur if hypertrophy co-exist,—that murmur actually is heard frequently under the circumstances,—and that, when wanting, its deficiency depends on the muscular energy being impaired by disease.* Hypertrophy with dilatation of a cavity, if its arterial outlet remain undilated, puts that outlet relatively in a state of coarctation: hence, too, may arise a murmur. I will return to these points with the subject of eccentric hypertrophy.

It was suggested by Martin-Solon that the pressure of the heart and great vessels by abundant pericardial effusion might cause murmur;—in a case of the kind, murmur, well marked in recumbency, disappeared when the patient stood up.†

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* Murmurs thus generated by *mis-direction of the blood-current* would really belong in the main to the dynamic species.

† Journal Hebdomadaire, ix. 457.

(*o*) The *properties of murmurs vary greatly with the conditions of the fundamental cause producing them*, and even with some conditions independent of this.

(1.) The force, loudness, or *intensity* of a murmur increases with the vicinity of its origin to the surface,—the density and hardness of the heart itself, and of the textures lying between the heart and surface,—the force and velocity of the current,—the amount of narrowing at an orifice,—and the volume of blood propelled through the obstruction. Excite a tranquil heart, and a murmur, previously, almost inaudible, becomes distinct; weaken the energy of cardiac contraction by tobacco or aconite, and the converse result follows.*

(2.) The *quality* varies with the character of the surface over which the blood passes;—harsh and rough, if the surface be sharply uneven; soft, if smooth and merely constricted. But this influence is greatly modified, and may be actually reversed, by changes in the velocity of the circulation. The quality will also be materially affected by the condition of the intervening structures: if these be soft, the sound will be softened in quality; if hard, hardened. A musical quality is sometimes given, when prominent spiculæ, or fibrinous particles, of vibratile character, project into the current; and also when rigid vibratile edges bound a narrow chink-like opening.

(3.) The *duration* of murmurs will increase directly as the extent of surface in the condition to afford them, the amount of difficulty to struggle against, the quantity of blood, and the slowness of the circulation. If the structures intervening between the seat of murmur and the surface be imperfect conductors, the audible sound will be shortened,—its termination will be lost through imperfect conduction.

(4.) The *pitch* of a murmur is more under the influence of the size of the orifice through which the sonorous stream passes, than anything else: the smaller the orifice, the higher the pitch. But it is also raised by the tension of the walls of that orifice, and the thinness of the blood. The velocity of the current does not influence it, Blakiston urges, unless, as in blowing with a flute, the harmonic be elicited. The opinion seems so reasonable that I almost hesitate to question its justness. Still, unless the edges of the diseased orifice be of such rigidity as to simulate the embouchure of the flute, it appears to me the two cases are not comparable; and as matter of actual experience I think it will be found a slight, though sensible, rise of pitch frequently takes place in murmurs when the circulation is excited.

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* In a woman now under observation (Emma Powell, U. C. H., Dec., 1850), the systoles are so unequal in force that while some give a strong radial pulse others produce none; the strong systoles are attended with systolic basic murmur, the weak ones with none.

The mere distance of the site of production of murmurs from the surface can in theory have no direct influence on their pitch. The same physical and dynamic conditions will generate sounds of the same pitch, whether they be close to, or as far as possible from, the chest-wall.

But may the pitch of a murmur change in course of conduction, —may the pitch differ, as perceived at different parts of the parietes of the chest? To whatever distance a note continues audible, it is commonly held, provided no intervening change of medium occur, to remain the same note, as at the spot of its generation.* However this be, it is certain that where any change of conducting medium occurs *in transitu*, a modification of pitch of the conducted sound will, or may, arise. If there be many such changes, occurring between the point of generation of the sound and the point where it is listened to, the varying effects of each on the pitch of the traversing sound, may by possibility correct each other in such manner, that the resultant at the end of the series does not perceptibly differ in pitch from the original tone.

Now these propositions are, as matter of experience, supported by clinical observation of cardiac murmurs; indeed, it was the observation of particular facts connected with the heart that led me to look after evidence to elucidate the general law.

Theoretically, change of the pitch of every individual murmur may occur according to the nature of the intervening media between the spot of production and of audition. Practically, such change is not often to be substantiated. For reasons not readily assignable, the murmur of aortic regurgitation furnishes the most marked and most frequent examples. Thus it is very common to find that murmur of higher pitch at the ensiform cartilage than directly over its site of production, the base of the heart; it rises by conduction. But the very same murmur, ausculted at the left apex of the heart, may have a different pitch from that of the other two spots,—lower somewhat than that of the neighbourhood of the ensiform cartilage, yet higher than that of the basic region. Difference in the conducting media in the two directions must be the cause of the difference in tone.[*]

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* I have long held and taught on the ground of experience, however strange the fact may appear, that if any reflection of sound occur *in transitu* from one spot to another, a change in pitch will ensue, though the conducting medium remain the same. And some experiments lately made by S. Ringer have led him to the conclusion that alteration of pitch occurs in tones conducted through one and the same medium,—that the ticking of a watch, for instance, rises in pitch the further it is heard in air from the point of its production, while with certain other kinds of medium a fall may take place.

I do not think the manner or degree of alteration in pitch are sufficiently uniform in occurrence to afford trustworthy indications in diagnosis.

(*p*) *Influence of palpitation on pre-existent murmurs.*—The influence of palpitation on already established murmurs varies. As a general rule, strong palpitation of regular rhythm intensifies the whole series,—the regurgitant quite as markedly as the direct class. I have, on the other hand, occasionally known a murmur, when audible in a calm state of the circulation, disappear during palpitation of rapid type,—but this only in some cases of mitral regurgitation. Can its cessation depend on irregular contraction of the wall of the ventricle allowing of such slight and feeble regurgitation that morbid sound cannot be generated? Again, in a case of enlarged, soft, flabby heart, with a congested state of the lungs and abdominal viscera, so long as the heart's action was in its habitual state of frequency and irregularity of rhythm, no murmur could be detected either in the erect or recumbent postures; but when the number of the pulse had been reduced by digitalis, a distinct murmur, systolic at the left apex, attended every beat of the heart.

(*q*) From various of the considerations into which we have now entered it would follow that the properties of a murmur, as caught by the ear, are every one of them, singly, of complex mechanism, and herein appears an easy clue to the absolute failure of all clinical attempts to establish the *precise amount and character* of the organic changes in a set of valves from the consideration of any one property, such as roughness, of the murmur they generate. Every necessary organic condition of a harsh murmur may be present, and yet the resulting murmur be soft, if the current be feeble,—nay, murmur may be wholly deficient.

(B) *Inorganic Endocardial murmurs.*—Endocardial murmurs that cannot be traced to any organic cause after death, or that disappear so completely and permanently during life, as to preclude the idea of structural change in their production, are termed inorganic. They are divisible into two sub-classes—murmurs originating in some unnatural condition of the blood (*hæmic*), or in perverted action of the heart (*dynamic*).

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[*] Flood, U. C. H., Males, vol. vii., p. 265, May, 1852. Case of highly marked aortic regurgitation: "the murmur tailed on to the second sound is higher in pitch at the ensiform cartilage than at the base . . . passing from the former point towards the left apex, it falls in pitch."

(1.) *Hæmic Intra-cardiac Murmurs.*—Murmurs originating within the heart and dependent on an unnatural state of the blood, are referrible to two species; in the one (*a*) the blood is altered in composition, but perfectly fluid; (*β*) in the other its composition is not demonstrably changed, but from some cause or other it has coagulated in the heart's cavities.

(*a*) *Hæmic murmurs depending on changed composition.*—An intra-cardiac hæmic murmur of this variety is of moderate or very slight intensity, commonly of medium or low pitch, short or moderately prolonged, of whiffing quality, very easily rendered temporarily harsh by excitement of the heart, and often modified in intensity by certain changes of posture. This murmur is, as far as I have observed, invariably basic in seat and systolic in time, produced at the orifices of the aorta and of the pulmonary artery,—with a force at each proportional to the power of its communicating ventricle; scarcely conducted in the direction of the aorta at all; frequently audible, on the contrary, at the second left, or pulmonary [19]* cartilage; only in exceptional cases audible below the nipple; and never, within my experience, perceptible as far as the left apex, or outwards towards the axilla.

It is *à priori* intelligible that a *diastolic* or *præsystolic* murmur at the left apex may be generated, independently of alteration of the mitral orifice, either statical or dynamic, essentially through morbid soniferousness of an anæmic blood-current, provided the auricle be hypertrophous and the flow of the fluid forcible to excess,—but I have never met with a satisfactory example of the fact.† But the doctrine of some observers, that true blood-murmur may be *systolic* in time and seated at the apex appears to me wholly untenable: how deficiency of red particles in the blood can lead to its reflux through the mitral orifice I am at a loss to conceive.

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* This clinical fact does not appear to me to prove that anæmic murmur is more commonly generated at the pulmonary than at the aortic orifice, but simply that the nature of the media lying in the direction of the pulmonary cartilage is more favourable to conduction.

† The intimate connection between hypertrophous left auricle and *organic* disease of the valve would of course interfere with the clinical discovery of the fact, admitting its reality.

Hence, it would follow, the site and rhythm of hæmic murmurs, excluding all those of diastolic time and as matter of experience of seat at either apex, are of great value in their distinction. To their quality and pitch I attach but moderate importance; for organic murmurs may be soft, and inorganic ones are not very unfrequently rather harsh; while the latter may be shrill, whistling, and of high pitch, and the former are of course frequently of low pitch. Unfortunately there is no character in a systolic basic blood-murmur which positively proves its nature, and distinguishes it, *under all circumstances*, from one of organic source. The distinction is often rather to be made through the absence or presence of venous hum (I do not remember ever to have observed an intracardiac spanæmic murmur unattended with venous hum), and the course and duration of the phenomenon, than through its own intrinsic characters: *permanent* harshness and high pitch are, however, never associated with true hæmic murmur. If arteries, on which the pressure of the stethoscope can have no influence, as, for example, the arch of the aorta, the innominate and sub-clavian arteries, be the seat, extensively, of strong blowing murmur, a co-existing systolic murmur at the mid-sternal base is, in part at least, inorganic; but, on the other hand, the cardiac murmur may be truly inorganic, and yet the arteries be perfectly free from abnormal sonorousness.

The morbid state of the blood most frequently associated with these murmurs is spanæmia,—whether it be that of chlorosis, of malaria, of starvation, of deficient insolation, of hæmorrhage and over-venesection, of carcinoma, of convalescence from acute diseases, &c. I have occasionally observed it in uræmia; so, again, it now and then occurs, though to a very slight amount, in the hypinosis of continued fever and the exanthemata, and in the hyperinosis of pneumonia and acute rheumatism, under circumstances excluding more or less positively its dependence on endocarditis. It has been affirmed that plethora, rendering the quantity of blood too great for the cavities of the heart, produces murmur within the organ: confirmation of the statement is wanting; more especially if we accept the teaching of those chemists who maintain that the relative quantity of red corpuscles is increased in plethora. Cyanæmia does not produce murmur.

The mechanism of this variety of hæmic murmurs will be considered with that of the venous class.

(β) *Hæmic murmurs depending on coagulation.*—These murmurs form a link between the inorganic and organic classes. Their varieties are exceedingly numerous. Here must in strictness be included all murmurs depending on fibrinous deposits on the endocardial surfaces, wart-like, stratiform, or other; and on similar accumulations in fissures of the valves or tendinous cords, or between the columnæ carneæ. In these cases, however, the process of deposition is slow,—the essential conditions involve, practically speaking, structural changes, and the attendant murmurs are unquestionably not to be clinically distinguished from those of ordinary organic mechanism. But to the present group belong also murmurs caused by sudden coagulation within the heart's cavities,—and to these only is the following description to be understood to refer.

These coagulation-murmurs are materially most common on the right side of the heart, simply because the tendency of the blood on that side to coagulate during life is greater than on the left. Clots are obstructive to the onward current,—these murmurs are consequently of direct mechanism, systolic when produced by clots in the ventricle. It is conceivable that a murmur of this mechanism may be of præsystolic or diastolic rhythm also, when clots accumulate in the auricle, or at the tricuspid orifice in such manner as to obstruct the onward current, or at the arterial orifices in such manner as to cause regurgitation. But I have not actually observed any of these conditions. The pitch of these murmurs is low or medium; and, commonly, soft, they are never, as far as I know, markedly harsh.

The circumstances under which intra-cardiac coagulation occurs, and under which murmurs may be looked for, are mentioned in the account of BLOOD-CONCRETIONS WITHIN THE HEART.

(2.) *Dynamic intra-cardiac murmurs.*—Under this head murmurs, occurring in the heart through abnormal action, range themselves.

(a) Violent excitement of the organ, whether it act merely by increasing the force of the current, or by disturbing its direction, occasionally produces for the time systolic murmur at the base. I have observed this not only in hysterical females, who, though of florid countenance, might, it is true, have been slightly spanæmic, but in males with palpitating heart. If the organ be the subject of dilated hypertrophy, palpitation is sometimes, but certainly not always, attended with the same murmur. In hypertrophy with dilatation there is possibly, we have seen, another dynamic source of systolic murmur: from the altered form of the cavities of the ventricles, their contained blood is probably propelled against the edges of the arterial orifices, instead of directly through them; and this misdirection of the current may, very possibly, generate murmur [p. 82].

(b) The heart may, probably, also undergo dynamic changes interfering with the closure of its valves, and giving rise to murmurs of the regurgitant class.[*]

Thus, a systolic murmur, free from harshness and of medium pitch, having all the attributes of that denoting mitral regurgitation (but invariably *unaccompanied* by accentuation of the diastolic sound at the second left, or pulmonary, cartilage), is occasionally heard in cases of chorea at the left apex. This murmur disappears as the primary neurotic disease itself disappears; its intensity varies with different beats of the heart; its quality similarly changes; and it may even temporarily become inaudible. The murmur in question is not localised as a hæmic cardiac murmur would be [p. 86]; instead of growing more marked, like a blood-murmur, as convalescence (with its attendant anæmia) advances, it lessens in intensity; it cannot be referred to organic change in the mitral valve, seeing that it eventually totally disappears. Nor in many of the cases which I have seen was there the smallest ground for referring it to latent Bright's disease, or rheumatic irritation of the endocardium.[†] The most plausible hypothesis seems to be, that irregular and occasional reflux takes place at the mitral orifice, through disordered action of the muscular apparatus connected with the valve.[‡] Just as the external choreal movements sometimes cease for moments or for minutes at a time, so the cardiac murmurs disappear. The main objection to this dynamic hypothesis seems to arise from the difficulty of understanding how the heart as a whole can act regularly (as it certainly does in chorea), and yet part of its substance contract irregularly. Still the same contradictory state of things may sometimes be witnessed in the voluntary muscles,—portions of which may twitch automatically while the remainder of their substance is quiescent. And as the physiological synchronism of contraction of various parts of the ventricle is ensured by the simultaneousness of influence of its various motor ganglia, it is quite intelligible that in a morbid state of things the necessary simultaneousness might be disturbed. Further, *portions* of the ventricle of the frog, poisoned by digitalis, contract spasmodically before others.

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[*] In the classical work of Stokes, an opinion is by inference ascribed to me on the subject of inorganic cardiac murmurs, differing from that I have really professed. Stokes seems to infer that I deny the possible production of systolic murmur at the apex, independently of textural disease of the mitral orifice (Diseases of the Heart, p. 496). Far from this, I have always strongly insisted on the probability that *dynamic* murmur of this site and rhythm really occurs; but cardiac murmur of *blood-origin* I have supposed, and still suppose, to be always basic in seat. I have never yet heard in a purely chlorotic woman a murmur having all the characters of a mitral regurgitant one. But these propositions are fully dwelt on in the text, p. 86.

[†] I may mention here, that my experience is far from supporting See and others as regards any necessarily intimate connection between rheumatism and chorea.

[‡] Among numerous illustrative cases may be selected: Robinson, U. C. H., Females, vol. xiv., p. 90; Gosling, U. C. H., Females, vol. xiv., p. 124.

Again, I have observed the same phenomenon of occasional systolic apex-murmur, lasting commonly for two or three days in connection of sequence to the fit in grave epilepsy. In one such case, that of a male, past sixty years of age, who remained under my tolerably constant observation for years, the manner of life was to the last degree antagonistic to the occurrence of anæmia. Besides, the blood must have been of precisely the same composition, when the murmur occurred, as when it was absent. I must add, the relationship to the fit was not always the same. Further, the systolic murmur at the left apex occasionally met with in masturbators, who are perfectly free from any of the acknowledged physical signs or symptoms of anæmia, and who have none of the habitual attendants of organic mitral reflux, appears to me to be of the same species.*

The same kind of disorder may conceivably be the cause of certain mitral regurgitant murmurs, attending dilated hypertrophy of the left ventricle, and disappearing under treatment.†

So, too, may be explained a fact, for which it seems otherwise impossible to suggest any plausible theory, that in certain cases of soft, flabby, and fattily atrophous heart, a murmur at the left apex will occasionally be heard, though as a rule completely absent.

May it be, that defect in the condition of the elastic tissue, which seems to have been shown by W. O. Markham to exist in the valves, allows of their eversion into the auricle? Or, again, is it possible that the innervation of the auriculo-ventricular valves may be disordered, so as to impede their closure? They are provided with sympathetic filaments; and (as is admitted to have been demonstrated by Senac and Kürschner) they enjoy a supply, though limited, of muscular fibre; why then, may they not palpitate as well, for instance, as the aorta?

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* Brereton, Brit. and For. Med. Chir. Rev., July, 1861, p. 266.

† Bonsey, U. C. H., Males, October, 1850.

I am aware there are some propositions pretty generally received by physiologists, concerning the part played by the papillary muscles, that appear hardly to warrant such importance being attached to a disordered state of their contractions. Thus, if the heart be removed from the body, an auricle cut away, the artery of the same side tied, and the cavity of the venticle filled with fluid, and if then a stream of water be directed upon the auriculo-ventricular valve, this rapidly closes. From this experiment of Baumgarten, the inference has been drawn that the auriculo-ventricular valves are closed by the systole of the auricles, prior to the systole of the ventricles, and that the closure is not in anywise influenced by the musculi papillares, but is much facilitated by the specific lightness of the valves themselves. It is further inferred that the papillary columns *merely afford* a *support to the chordæ tendineæ*, which in turn prevent the valve from floating too far on the auricular side. The multitude of points in which the experiment fails to imitate the natural state of things in the living and contracting heart, gravely invalidates, in my opinion, the conclusion it has been forced to furnish. There is in truth the strongest reason for believing that the papillary muscles by their contraction draw the valves somewhat away from, as well as prevent eversion into, the interior of the auricle.* But, even granting that the inference in question be sound in *physiology*, the additional assumption of J. Hamernjk, that *morbid* conditions of the muscular structure of the heart can have no effect in preventing closure of the valves, seems a palpable *non sequitur*. What! suppose that (*inter alia similia*) the papillary muscles are shortened, puckered, dwindled in muscular texture, and infiltrated with pseudo-fibrous induration-matter, in a word cirrhosed, will not this state somewhat, at least, interfere with the function of the valves? Why, this would almost amount to affirming that shortening and thickening of the chordæ tendineæ can produce no embarrassment in the play of the valves.†

I have met with some few instances of inconstant murmur-like quality of the diastolic sound at the base, which appeared to me to be possibly dependent on disordered dynamism of the aortic valves. Are there muscular or nervous elements within them?

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* This experiment, which is easy of execution, proves, however, that W. O. Markham (Med. Chir. Trans., vol xliv.) cannot be right in affirming that "the stream of blood flowing from the auricle into the ventricle cannot possibly raise the valves upwards."

† In an experiment with digitalis on a dog a temporary murmur was observed by Brunton and Gamgee, which they referred to reflux, caused by irregular action of the papillary muscles. (Fothergill on Digitalis, p. 7, 1871.)

In certain cases of apparently purely dynamic palpitation of excessively irregular rhythm and force, murmur-like quality, or actual murmur, may be momentarily heard—now basic, now at the apex, now systolic, now diastolic. These anomalous conditions find their explanation in the foregoing statements. But I must add that I have not actually noticed *well-defined* murmur, constant for any length of time, unless there were, on other grounds, more or less strong reason for the suspicion at least of organic disease.

107. *Of Individual Endocardial Murmurs.*—In endeavouring to ascertain the seat of production of any given endocardial murmur, the essential points in the inquiry are its relationship to the systole or diastole, and the spot of its maximum intensity on the surface of the chest. Subsidiary conditions of great importance are the direction of transmitted audibleness, duration, clinical progress, quality, and pitch of the murmur; while, in addition to its own characters, the state of the heart's natural sounds, and the presence or absence of certain audible phenomena in the arteries and veins, or both, are valuable subsidiary guides.

108. Each orifice of the heart may be the seat of two murmurs, obstructive and regurgitant,—with, or against, the current: the total number of cardiac murmurs directly connected with the orifices, therefore, reaches eight. The essential characters of these may be briefly set down as follows:—

109. (*a*) *Systolic Murmur of the left apex.*—A *systolic* murmur of maximum force at and immediately above, or to the outside of *the left apex;*[*] but faintly audible, or wholly inaudible, at the right apex (say, the ensiform cartilage), the mid-sternal base,[†] and the pulmonary and aortic cartilages; more or less clearly audible about and within the inferior angle of the left scapula, and in the left vertebral groove from the sixth to the ninth dorsal vertebræ,[‡] audible or not round the lateral base of the chest from the cardiac to the scapular region, is essentially characteristic of *regurgitation through*

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[*] Occasionally a case presents itself in which a murmur of mitral reflux mechanism (as proved *post-mortem*) is audible with greatest distinctness *above* and somewhat outside the nipple. I have not succeeded in finding any anatomical explanation of this anomaly.

[†] If the stethoscope be carried gradually upwards and inwards from the maximum point towards the third cartilage, it will in many, but not all, instances be found that at a certain line, defined with singular sharpness, the characters of the murmur completely change: in intensity, it falls to a third; in roughness, loses greatly; in pitch, commonly falls.

[‡] I have occasionally known a murmur of indubitable organic mitral mechanism better audible at the upper than the lower part of the left vertebral groove, and con-

the mitral orifice at the moment of the ventricular systole. This regurgitation may be the result of inefficiency of the valve, produced by chronic changes of structure (its common cause),—or by enlargement of the orifice without coeval growth of the valve (a *very* rare cause),—or occur from non-closure in acute endocarditis, in consequence of roughness of the edges of the valves, ill-fitting opposed elevations on their surfaces, and shortening of the chordæ tendineæ.

Of very variable quality, this murmur is rarely of high pitch, generally oscillating between whispered *who* and *rr;* but I have known it sharply whistling, as of the *wind through a key-hole;* it is occasionally distinctly musical. Once established, it is, as a rule, permanent; but when, probably, dynamic, as in chorea, epilepsy, &c., it may momentarily, as well as permanently, disappear. If the cause of non-occlusion, even though it be organic, act intermittently, the murmur will be present at some moments, absent at others. This curious peculiarity I once observed in an adult male, in whom a body about the size of a large pea was suspended by a thread-like peduncle from the larger division of the valve, in such manner that it might or might not, according to accidental circumstances, have fallen within the orifice, and impeded its closure.

A mitral regurgitant murmur may completely or partially cover the first sound at the left apex. This first sound may have its natural characters in perfection at the base and at the tricuspid apex; but when intense the murmur may partially obscure the sound in both these places by conduction.

In the healthy state, the second sound is more strongly accentuated in the aorta than in the pulmonary artery. The reverse, according to Skoda, holds in cases, among others, of mitral regurgitation; and the peculiarity is explicable thus:—With every systole some blood is forced into the left auricle; that auricle, the pulmonary veins, and pulmonary artery quickly become overstretched, and the right heart requires greater effort to force the blood into the over-filled vessels; the pulmonary artery consequently presses with increased force on its column of blood, and hence intensifies its own portion of the second sound. And, as a corollary, he holds that the absence or presence of this reinforcement will distinguish systolic murmur

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sequently in so far simulating an aortic murmur. This must depend on some unusual condition of the lung and other intervening conducting material. (Dunn. U. C. H., Females, vol. xvi., p. 296.) The murmur may even be *inaudible* at the inferior thoracic part of the groove, while distinct superiorly,—*e. g.* Mahon, U. C. H., Females, vol. xv., p. 134. For the topography of the valve posteriorly, vide [34].

at the left apex, caused by regurgitation, from that caused by friction of the blood against roughnesses in the ventricle. I do not believe, although it certainly does exist in some cases, that any such implicit confidence can be placed in this sign. I have known it wanting in cases of mitral regurgitation, even when there was no obvious tricuspid regurgitation to afford a plausible explanation, through diminished amount of blood-current, of its deficiency.* It seems to be sometimes, too, only a pseudo-accentuation of the pulmonary second sound, from real weakening of the aortic second sound, arising through the lessened current and dimished calibre of that vessel, that follow on long-continued mitral regurgitation. Besides, I have not found thickening or enlargement of the pulmonary artery in such connection with mitral regurgitation, as ought to obtain, were the theory described wholly accurate,—the more so, as thickening and enlargement of the left auricle really do exist in a proportion of these cases. Hypertrophy of the right ventricle even *may* be present without entailing any intensification of the second pulmonary sound.

Mitral regurgitant murmur may be highly marked with strong, deficient with feeble, systoles.†

A murmur, possessing *all* the characters just enumerated, may lay claim to indicating, with well-nigh absolute surety, the existance of *mitral reflux of organic mechanism.* To have such significance must it actually possess the entire group? This is still to a certain degree a moot question, in regard of one of the most important of the whole series, audibleness within the inferior angle of the scapula. For my own part, I feel persuaded such audibleness (to be substantiated in about two of every three cases) is less a measure of intensity than an index of mechanism. If the murmur be imperceptible posteriorly, I believe it *very seldom depends on regurgitation of structural mechanism;* possibly it may *never* do so, but I am unprepared to adopt so sweeping a formula. Still, I think, we may say it *never* depends on *well-pronounced organic* regurgitation.‡ On the other hand, systolic apex-murmur inaudible posteriorly, is the exponent of dynamic mitral reflux, or of various conditions, unnaturally soniferous, occurring in the interior of the ventricle. In cases of chronic abiding murmurs the deficient audibleness behind is rarer than in those of acute mechanism.

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* In a valuable table of 100 cases of systolic apex-murmur collected by J. Andrew, the absence of intensification of the diastolic sound at the pulmonary cartilage is noted 30 times. It is true, however, we do not know in how many of the whole series the murmur depended on actual reflux. (Bartholomew's Hospital Reports, vol. i., p. 16, 1865.)

† Allen, U. C. H., Females, vol. xvi., p. 167.

‡ Besides, as just shown (*vide* note, p. 92), organic mitral murmur may not occupy its habitual site in the groove.

A murmur, audible at once in front and in the left vertebral groove, may be traceable continuously round the left lateral base of the chest, or it may be lost for a greater or less part of the distance between the two points. The difference seems unconnected with mechanism, and really referrible either to varying intensity of the murmur, or to some accidental peculiarity of the conducting media.

Further, if the description above given of accentuation of the pulmonary second sound be correct, it follows that, as far as my information goes, its *presence* cannot be held to *prove*, or its *absence* to *disprove*, with perfect security, the structural and reflux mechanism of a systolic left-apex murmur.

We may then accept for clinical purposes the following arrangement:—

SYSTOLIC LEFT-APEX MURMUR.

	Seat of production.	Cause.	Mechanism.
Audible in left vertebral groove.	Mitral orifice.	Structural insufficiency of valve.	Reflux through orifice, often with roughened edges.
Inaudible in left vertebral groove.	Mitral orifice.	Dynamic interference with closure of valve.	Reflux through orifice in itself smooth and natural.
	Interior of ventricle.	Roughness of endocardium of ventricle, or of inferior orifice of valve.	Abnormal collision of blood.
		Mis-attachment of a tendinous cord.	
		Thickening and roughness of cords.	
		Fibrinous shreds crossing line of current.	
		Coagula in cavity.	
		Sacculated aneurism of wall of ventricle.	Egress of blood from sac at moment of systolic contraction.

110. (β) *Systolic Murmur at the right apex.*—A *systolic murmur* of maximum force *immediately above, or at, the ensiform cartilage;*

inaudible, or nearly so, at the left apex; and, according to my experience, inperceptible in the left vertebral groove opposite the lower angle of the scapula, originates in connection with the *right ventricle.* In the great majority of cases this murmur arises from *tricuspid regurgitation.* It may also by possibility depend, at least theoretically, on sharp collision of blood among the thickened and roughened chordæ tendineæ.[*] In the former case, distension and pulsation of the auricle, vena cava, innominate and jugular veins is habitually present, that of the latter *visible* but not *palpable;* but the absence of such pulsation does not prove that the murmur under consideration is independent of regurgitation.[†]

This tricuspid murmur is generally soft, and of low pitch, rarely masks the systolic sound completely, is of rarer occurrence than tricuspid regurgitation itself, and is probably not always detected when it exists. It is, absolutely speaking, rare, because regurgitation often occurs from insufficiency, without roughness or other morbid change, of the valves, and because the back current is often not forcible enough for the production of a murmur. On the other hand, it escapes detection, because it is often covered by a powerful mitral murmur, and in some cases impaired in distinctness by *deep-seated venous hum.* In rare, very rare, instances, where a mitral and tricuspid murmur coexist, a spot may be found midway between their points of maximum force, where there is comparatively little murmur.

In certain cases of mitral regurgitation, the attendant murmur may be so deadened at the left apex by emphysematous lung, as to be better audible at the right apex, and so simulate a tricuspid murmur.[‡]

111. (γ) *Systolic basic Murmur transmitted to the right and upwards.*—A *systolic murmur*, of maximum force, *at midsternum, opposite the third interspace* (or, it may be, the upper part of the fourth rib); gradually or abruptly losing force between this point and the apex-beat, where it may be almost inaudible; faintly perceptible at the second left cartilage; clearly audible at the second right cartilage, the notch of the sternum, and the left vertebral

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[*] I have never heard a murmur indisputably proved after death to have been thus generated. Nor, although Skoda and others admit without any apparent hesitation this form of mechanism (Skoda, Markham's Translation, p. 236), I know of no printed clinical demonstration of the fact. At the same time, I do not contest its possibility, especially if the ventricle be hypertrophous and dilated.

[†] Hishin, U. C. H., Females, vol. xi., p. 288; well-marked tricuspid regurgitant murmur, and no actual jugular pulsation.

[‡] Hutchinson, U. C. H., Males, vol. viii., p. 335.

groove, opposite the second, third, and fourth vertebræ, thence rapidly losing strength downwards, and disappearing about the sixth, originates at (and synchronises precisely with the passage of the current through) the *aortic orifice.*

The combination of these characters gives distinctness, beyond cavil, to this murmur. But a direct murmur through the aortic orifice may differ in some respects from this typical picture. Thus, when the heart hangs low, and the organ lies more horizontally in the chest, than is habitual, this aortic murmur may have its maximum-point of intensity much lower than usual. I have known that point fall almost as low as the articulation of the sternum with its appendix,—when obviously the murmur might (unless from its intensity and the nature of its accompaniments, positively and negatively considered) simulate a tricuspid reflux murmur. Again, in some instances, in consequence of the relationship of the vessel to the surface and the nature of the intervening material, this murmur is better conducted to the *left* than to the *right* of the sternum in an upward direction [19]; and, per contra, it may be better audible down the sternum than in the direction of the left ventricle. Again, it may be more distinct in the upper part of the back to the right than the left of the spinal column,—the lungs, mediastina, and arch of the aorta being, as far as evidence goes, healthy; or it *may be wholly inaudible in the back.*

This murmur is commonly of high pitch, loud, prolonged, and harsh; it may, however, be low-toned, and of peculiar twanging, musical quality.[*] If the ventricle behind be dilated and hypertrophous, this state of things increases its intensity, and may give it a drawling, prolonged character, if the sides of the orifice be much contracted.

Although well audible at the aortic cartilage, it is distinctly fainter there than at the base; if as marked at that spot as, and *à fortiori* if more marked there than, opposite the valves themselves, disease of the arch of the aorta itself adds to the murmur.[†] The same inference is justified by any *notable* difference in pitch at the two spots. Obviously such amount of change, either in strength or pitch, could not be the effect of conduction.

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[*] Gall, U. C. H., Males, vol. xvii. p. 272.

[†] There is one kind of exception to this statement: if the apex of the right lung be solid, the murmur may be more marked at the aortic cartilage than at the base, though the aorta is healthy. I have never known more than slight excess under these circumstances [100].

The conditions, generating a murmur of the attributes now enumerated, may be tabulated as follows :—

SYSTOLIC BASIC MURMUR CONDUCTED TO THE RIGHT.

NATURE	SEAT OF PRODUCTION	CAUSE
Structural.	Aortic orifice.	Stenosis, rough or smooth. Fibrinous coagula obstructing orifice.
Dynamic.	Aortic orifice.	Violent excitement of heart. Mis-direction of current against sides of orifice.
Hæmic	Aortic orifice.	Spanæmia. Uræmia.

SIMULATING MURMURS.

Structural.	In left ventricle.	Sacculated aneurism of wall of ventricle.
	Above heart's base.	Pouching of aorta close to sinuses of Valsalva. Varicose aneurism of arch.
	About first right interspace close to sternum.	Coarctation of arch of aorta.
	Either right or left second interspace.	Embrace by pericarditic lymph or pressure by fluid on roots of great vessels. [135.]

A glance at this table will at once convince the student how erroneous it would be to suppose the aortic valves of necessity diseased because the systole was accompanied by murmur of the character described. The inference would be unwarrantable, both because other conditions generate true basic systolic murmur of the kind,—and because a simulation of the murmur is produced by several other diseases. The means of diagnosis will be fully considered with the descriptions of the diseases concerned,—but it may meanwhile be frankly admitted here, that in some of the cases of simulated basic murmur, error is escaped with difficulty. It is easy to say some of these simulated murmurs are slightly præ-systolic in time, others slightly post-systolic; but to feel certain at the bed-side concerning divisions of time so minute is often beyond our power. The real safety against error lies mainly in the rarity of these causes of the simulations in question.

112. (δ) *Systolic basic Murmur transmitted to the left and upwards.*—A *systolic murmur*, of maximum force *at the sternal edge of the third left cartilage*, or a little lower down, loud at the pulmonary cartilage, notably less audible at the aortic cartilage, perceptible (more or less markedly according to the loudness at its

point of maximum force) at the upper part of the front chest, but faintly transmitted towards the apex, inaudible in the carotids, and imperceptible, or almost so, in the back, originates within, or close to, the orifice of the pulmonary artery.

Structurally this murmur depends on stenosis of the orifice of the vessel, or roughness of its valves; or, with infinite rarity, as noticed by Elliotson, on pressure of the vessel, just above the valves, by adventitious material in the pericardial sac.

If we except cases of cyanosis (where stenosis of the pulmonary orifice is the most common lesion), the conditions capable of generating this murmur are so rare, that few persons have actually met with an example. Setting aside cases of cyanosis in infancy and childhood, I can recall in my own experience only one well-defined instance of murmur of this site, time, and mode of transmission; and as in this case there was no sectio cadaveris, its site cannot be held to have been established with certainty, so limited is our experience of its characters.

As already mentioned [19], it does not appear that the transmission of an anæmic murmur, from the base, rather to the left than the right of the sternum, indicates that the sign is really generated at the pulmonary instead of the aortic orifice. We have to deal with a phenomenon of conduction allied in nature to that which habitually renders an aortic reflux murmur better audible at the ensiform cartilage than, as might be expected, at the apex of the heart. But it must be admitted the fact, which is unquestionable, introduces an element of confusion into the diagnosis.

113. (ε) *Præ-systolic or post-diastolic murmur at the left apex.*—A murmur commencing at the close of, or instantly after, the ventricular diastole; synchronising precisely with neither sound, but following the second and anticipating the first, which closes it more or less sharply at the end of the long pause; of maximum force immediately above and about the left apex; very rarely audible higher than the fourth interspace, and conducted in the same directions as, but much less effectively than, systolic murmur of the same seat, and comparatively seldom audible in the vertebral groove, though occasionally to be heard over the entire back, indicates *obstructive narrowing of the mitral orifice*, or, possibly, *simple roughness of the auricular surface of the valve.*

This murmur, which, as I have invariably said in all editions of this work, including the first of 1851 (p. 226), is in rhythm "rather post-diastolic or præ-systolic than precisely coincident with the diastole," has, until of late, been habitually spoken of as "diastolic." Probably writers have fallen into this error from the fear of being taxed with over-refinement. For error it certainly is, as shown so long ago as 1843 by Fauvre,* who either invented or adopted the term præsystolic to designate its rhythm with precision. W. T. Gairdner, in the course of a lucid exposition of cardiac murmurs, names it "auricular systolic," combining at once the ideas of mechanism and time.†

If its usual rhythm alone be considered, this murmur may be styled indifferently *præsystolic* or *post-diastolic*,—but the former term seems the best, because implying, as is the fact, that the murmur is not tailed on to, but marks at the moment of its occurrence the initiation of each of the heart's revolutions it accompanies. Besides, it must be confessed, the precise synchronism of the murmur is often very difficult of establishment: the rule already laid down for timing apex-murmurs in general [106, *j*] must be carefully applied. I am, for my own part, satisfied from observation that the murmur in some cases commences synchronously with, and not after, the close of the diastole.

Præsystolic murmur, of variable but considerable intensity, ranges between low and medium pitch, is commonly prolonged,‡ and of more or less coarse quality, often harsh, rough, or even grating, and seldom, if ever, soft as an enduring condition.

The habitual inaudibleness of this murmur in and about the vertebral groove, as compared with the audibleness of structural reflux murmur at the same orifice, may probably depend on the difference in the direction of the two blood-currents concerned,—in other words, upon convection, *from* the back in the former case, *towards* the back in the latter. The prolongation of the murmur will, in all likelihood, vary directly both as the length of the funnel-shaped opening into which the contracted orifice is converted, and as the amount of dilatation of the auricle, which regulates the quantity of blood to be projected through the orifice.

It has not occurred to me to observe any case in which mere roughness of a mitral orifice of natural width has given rise to murmur. Skoda, however, not only speaks of its production by such unassisted roughness, but affirms that murmur of this mechanism may be distinguished from the constrictive variety by the condition of the second sound in the pulmonary artery,—this sound will be loud to excess in the case of narrowing; unaffected in that of simple roughness, unless other causes of pulmonary arterial reinforcement be present. The distinction will, however, if well-founded, be clinically useful only in cases where the absence of mitral regurgitation and of right ventricular hypertrophy is certain.

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* Archives Gén. de Médecine, 1843.

† Clinical Medicine, 1862.

‡ C. Hilton Fagge (Guy's Hosp. Rep., 1871) infers from an observed case, that "the murmur is occasionally so short and sudden that it resembles rather a tone, and is in itself hardly to be distinguished from the natural first sound of the heart."

Præsystolic apex-murmur is not very unfrequently wanting, where mitral constriction is found after death. Sometimes the deficiency may be fairly referred to the weakness of the auricular systole and smoothness of the constricted orifice;[*] where the constriction is slight, the friction will also be but slight. When deficient, as it has positively been, in cases of marked contraction, Hope thought the deficiency depended on the very fact of the extreme smallness,—an explanation not over plausible. The plain cases, in which *præ-mortem* exhaustion has probably prevented a murmur being heard in persons, seen only within a day or two of death, do not require special consideration.

I have known this murmur come and go from day to day in a case where the mitral orifice was very greatly contracted and rigid:[†] probably from varying force of the heart's action. Henry Simpson has confirmed this observation: in one of three cases, recorded by him, the murmur changed from a state of harsh distinctness to almost total extinction in the course of a few minutes.[‡] In such cases the explanation is not easily found.

Such, succinctly stated, are the characters of the murmur attending mitral stenosis. Concerning these characters little difference of opinion can arise. But the mode of production of the murmur, as commonly admitted, has of late been questioned by A. W. Barclay, who insists, in an argument of great ingenuity, that instead of being direct or obstructive, the mechanism is in reality regurgitant.[§] A. W. Barclay maintains that a period of ventricular action, occurring principally at the base, follows the auricular systole, precedes the actual apex-beat, and gives rise to reflux before completion of the ventricular systole. But this separation of ventricular systole into a pair of acts (one of them slow, for the murmur is often prolonged) seems scarcely possible: all physiologists insist on the suddenness, quickness, and uniformity of distribution of action of the ventricle in warm-blooded animals during systole. There are many other reasons for declining to accept this doctrine of regurgitant mechanism—reasons urged with singular force by G. W. Balfour* in a logical analysis of Barclay's paper. Meanwhile, pending the settlement of this very difficult question of mechanism, it is satisfactory to know that the diagnostic significance of the murmur remains unshaken.

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[*] In the case of Denham (U. C. H., Males, vol. vi., p. 77, Feb. 1851), the long tongue of the mitral valve was much thickened; at its union with the other tongue it was puckered; and a mass of ossiform substance, as large as a very small marble, smooth on the auricular, rough on the ventricular surface, had formed at the union of the two tongues. Here there was no mitral constrictive murmur.

[†] Kernis, U. C. H., Females, vol. ii. p. 240, March, 1847. The auricle here was dilated, the endocardium "very thick and dense," but the muscular substance not markedly hypertrophous.

[‡] Brit. Med. Journal, May 16, 1868.

[§] Lancet, May, 1872.

113*. Præsystolic murmur is a *conceivable* effect of blood-struggle at an open foramen ovale; but such murmur would be of maximum force at and above the heart's base, and be almost inaudible at the apex [691, *f*].

114. (ζ) *Diastolic Murmur at the right apex.*—A *diastolic*, or, more accurately, *præsystolic murmur*, of maximum force, at *the ensiform cartilage*, most faintly audible at the left apex, and inaudible at the base, would probably indicate *tricuspid narrowing*, were there a hypertrophous auricle behind that orifice, to give force to the current. Experience concerning this murmur is on a very limited scale: the little I know of it by observation will be found in the description of Diseases of the Orifices [623].

115. (η) *Diastolic basic Murmur conducted to the right upwards and directly downwards.*—A *diastolic murmur*, of maximum force *at mid-sternum*, *the third interspace or fourth cartilage*, conducted (with some exceptions, to be mentioned presently) on the same principle as the systolic murmur of the same site, originates at the mouth of the aorta, and indicates regurgitation, or reflux, of the blood-current from the artery into the communicating ventricle.

This murmur may be heard with almost as much intensity about the ensiform cartilage as opposite the third interspace—in this point of view differing materially from constrictive aortic murmur: the more distant conveyance downwards in the former case probably depends on the downward direction of the current producing the murmur. So marked is the fact, that unless with care the murmur of aortic regurgitation might be mistaken for one of tricuspid constriction. I have even known the murmur louder at the upper edge of the xyphoid cartilage than at the base of the heart.† It is remarkable that the conduction is more perfect towards the right than the left apex,—consequently not, as might have been supposed, in the direction of the communicating ventricle: concerning this point I find my experience at variance with the statement of Hope. The murmur may, though well marked in front, be quite inaudible in the vertebral groove. The murmur is in rare cases louder to the left than the right of the sternum on the level of the second cartilages,—a special effect of conduction [19].

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* Lancet, May 25, 1872.

† Flood, U. C. H., Males, vol. vii., pp. 265, 266.

The second sound of the heart may be covered completely at the maximum point of the murmur; or it may be heard at the beginning of, during, or at the close of this. In the first case, the valves are utterly incompetent: in the varieties of the second case, one division of the valve may flap naturally, or all three imperfectly, and so allow of the production of an imperfect second sound; or the second sound heard may be wholly that of the pulmonary valves.

The most common of these compound conditions of diastolic murmur and sound at the base is that of murmur abruptly brought to a close by sound; a state pretty accurately rendered by the whispered symbols *phwi...tt* or *phwe...tt*,—the *tt* being sharply accentuated. Skoda thinks, under these circumstances, the murmur is due to friction of the recoiling blood during the arterial systole against the roughened coats of the aorta, and the sound to the natural closure of the valves without regurgitation. This explanation may hold in some instances; I am certain it will not apply to all. For I have observed this state of basic diastolic sound, where the existence of marked visibleness of all the superficial pulses left no doubt in my mind of the existence of aortic regurgitation,—the patients being besides young and free from notable hypertrophy of the left ventricle. In such cases the explanation may then be that partial aortic reflux takes place, the valves being partially, not wholly, disabled from closing. Or it may be that no diastolic sound is produced at the aortic orifice at all, only feeble murmur,—while the sound heard is that of the natural flap of the pulmonary valves, a little sequential in time: but the impression on the ear is so strong of the two divisions of the *phwe...tt* sound being produced at the same spot, and being distinctly connected with each other, that it is difficult to accept this explanation.*

To return to ordinary diastolic murmur: unless the murmur be of very great intensity, the second heart-sound may be well heard at the left apex: it may even be strongly accentuated there,*—a fact which has already [98] been made the subject of comment. The state of the second heart-sound at and about the second left, or pulmonary, cartilage, varies: it may be well or feebly audible, or wholly imperceptible,—a variation to be explained by the intensity and mode of conduction of the murmur in different cases.

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* In one case of the kind (Monk, U.C.H., Males, vol. xii., pp. 72, 86,) there was well marked reticulation of the aortic valves. It is, perhaps, conceivable that this condition might allow of reflux at the *commencement* of the time of closure of the valve.

Aortic regurgitant murmur is usually of blowing quality, sometimes almost hissing, generally of low pitch, rarely rough, weak as a rule (though I have known it extremely loud, and it is said to have been heard at a little distance from the surface), now and then of twanging, almost musical, character.

This murmur occasionally seems to pass *from* the direction of the ausculting ear: this will be the case, whenever the stethoscope happens to be placed higher than the heart's base. As a rule it is well prolonged, habitually filling the post-diastolic silence.

The causes of this murmur, or rather of the reflux of the current from the artery which produces it, are the ordinary morbid changes entailing incapacity of valves; I have known it caused by sudden rupture of one of the sygmoid valves.† Reticulation of the valves carried to extremes, a conceivable source of murmur, has never fallen under my notice as its cause (vide VALVULAR ATROPHY); but perforative destruction on a large scale will surely generate it. Mere insufficiency of valves, healthy in themselves, but too small to fill the morbidly widened mouth of the aorta, has in rare instances produced this murmur. In exceptional cases it is developed during the *acute* period of endocarditis.

It remains for future inquiries to determine, also, whether temporary reflux with its murmur may not be caused by dynamic imperfection without structural change in the valves.

116. (θ) *Diastolic basic Murmur conducted to the left upwards.*—From experiments on animals, it has long been known that a *diastolic* soft prolonged murmur, audible to the left of the sternum above the heart, and conducted down the right ventricle, may be produced by artificially rendering the *pulmonary valves insufficient.* But this regurgitant murmur is of excessive rarity in man; and the few recorded examples have, as a rule, undergone but slight clinical examination. As already mentioned, we occasionally find an aortic regurgitant murmur more distinct at the second left than the second right cartilage—an obvious source of fallacy.

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* Flood, U. C. H., Males, vol. vii., p. 265.
† Gordelier, Consumption Hospital, Males, vol. i.

And, in point of fact, the diagnosis of pulmonary reflux murmur is rather to be made with surety through the absence or presence of certain concomitant conditions than by means of its own intrinsic properties.

117. *Relative frequency and modes of association of endocardial organic Murmurs.*—In respect of relative frequency, I should be disposed to place intra-cardiac murmurs of *organic* origin in the following order, commencing with the most common :—mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive.

These murmurs may be variously associated; the following combinations are arranged in the order of their relative frequency: —aortic constrictive and mitral regurgitant; aortic constrictive and regurgitant; mitral regurgitant and aortic regurgitant; mitral regurgitant, aortic constrictive and regurgitant; mitral regurgitant and obstructive; mitral regurgitant and tricuspid regurgitant; mitral regurgitant and constrictive, aortic constrictive and tricuspid regurgitant; tricuspid constrictive and mitral constrictive.*

The existence of any one organic murmur does not involve, as matter of necessity, the presence of any other; a direct murmur may exist at any valve, and an indirect be absent, and *vice versâ*.

118. When two murmurs co-exist at the same orifice, they are, as a rule, readily distinguishable by their rhythm, their quality, their pitch, and by their appearing to pass towards, or from, the ear. I cannot believe with Skoda, that mitral systolic and diastolic (or better præsystolic) murmurs, forming one quasi-continuous noise, shall be in nowise distinguishable from a single murmur, until slackened action of the heart distinctly separates them into two.

119. *Substitution of one Murmur for another.*—(*a*). A given orifice may be the seat of an organic murmur of a given synchronism, which shall wholly disappear and give place to another of perfectly different relationship to the heart's rhythm. I first noticed this conversion at the aortic base in 1860,—a diastolic murmur, totally disappearing from that site, gave place to a systolic one. The simple and satisfactory explanation of this seeming paradox will be found with the description of DISEASES OF THE

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* The anatomical conditions of this latter singular combination are exemplified by Carswell's Drawings, U. C. Museum, Portfolio A, No. 285.

ORIFICES [636]. I believe I have observed the same phenomenon at the mitral orifice, but have no post-mortem evidence to offer.

(*b*) Certain diseases of valves may act now as the cause of regurgitation, now as that of obstruction, functionally considered. In a remarkable case of the kind, where the time of a murmur at and above the left apex had during life puzzled exceedingly those who watched the case from day to day, in as much as that murmur appeared purely systolic at one time, at another post-systolic, and again systolic and diastolic both, the explanation clearly arose out of the state of the mitral valve. The long division of that valve exhibited a rounded perforation, as large as a good-sized pea, closed up by a soft fibrinous vegetation, attached so loosely, however, to the edge of the opening, that the two things—perforation and plug—must have occasionally changed their relationships in such manner that either obstruction to, or regurgitation of, the blood-current, or both, must have been the passing results.*

Hence this kind of murmur-substitution may be a persistent or a temporary state.

B.—EXO- OR PERI-CARDIAL MURMURS.

120. Pericardial murmurs are divisible, in respect of quality and of the mechanism of their production, into four chief species: (*a*) Friction or attrition murmurs; (*b*) continuous murmurs; (*c*) clicking murmurs; (*d*) murmurs produced by bending of layers of exudation-matter. Roughness of surface is the essential statistical condition of the three first,—movement the essential dynamic element of all four.

(*a*) Attrition-murmurs, all of them, more or less, distinctly suggestive of rubbing of surfaces of variable character against each other, occur in a greater number of varieties even than pleural friction sounds. They resemble, for instance, *grazing, coarsely rubbing, grating, scratching, creaking, squeaking, and prolonged whistling* noises. Traceable as all these varieties commonly are to collision of surfaces roughened with lymph, any interest attached to their distinction must turn mainly on their being severally connected with some particular state of that lymph. Now, experience does not show any such necessary connection; though it be true, for the most part, that at the very earliest period the murmur is of grazing quality, like the sound produced by rubbing pieces of silk together, and that as the exudation hardens, and gathers into irregular peaked elevations, the quality becomes coarsely rubbing, grating, creaking, still this sequence of changes is subject to constant exceptions. On the one hand creaking friction-sound, so loud as to be audible *three inches from the auricular end of the stethoscope applied to the surface in the ordinary way*, may depend, I know from actual observation, on tough exudation-matter with fine rough elevations; and on the other I have noticed distinct, though slight, creaking quality, when the exudation-matter was found to be of almost creamy softness.* Taylor,† too, has known signs of moderate liquid effusion coexist with friction-sound of this quality,—a fact showing that peculiarly rough attrition is not required for its production.

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* Goodwin, U. C. H., Males, vol. xi., pp. 41, 85; further illustrated clinically by case of Williams, U. C. H., Males, vol. xvi., p. 10.

(*b*) When exudation-matter and fluid coexist, the former imprisoning the latter in its meshes, the heart's action may produce a peculiar continuous rumbling, or squashy churning sound, just such, as we find, occasionally occurs in the pleura. This is rare; but, once heard, can never be forgotten.

(*c*) Occasionally sounds are caught of peculiar clicking character, one or two, with each beat of the heart, which are only distinguishable, at the time, from modifications of the valvular sounds, by their non-synchronism with these, and by the extreme irregularity of their occurrence. I have satisfactorily traced these clicks to the pericardium, and further, in all probability, to the separation, without attrition, of surfaces glued together with exudation-matter.‡

(*d*) It has appeared to me that sound is sometimes generated in layers of firm exudation-matter, though so perfectly agglutinated together, that attrition or separation of the apposed surfaces would appear physically impossible. The quality under such circumstances is probably variable: in the only positive instance of the kind, which I have observed, it was faintly creaking. The bending and crumpling of tough exudation-matter may conceivably generate such sound.

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* "A murmur like that produced by rapidly rubbing the finger along a pane of glass, short and long alternately, very loud, not synchronizing with either heart-sound (both of which are natural) varying in intensity with position, louder in the sitting attitude than in decumbency, intensified by leaning forwards, and by holding the breath, of maximum strength about the left apex, wholly absent with some beats of the heart, and *audible from two to three feet from the surface of the chest.*" This observation was made on a female, aged 41; the murmur was evidently pericardial, and there was evidence to show it had existed for some considerable time [245, *d*]. But the precise state of the membrane giving rise to it is, of course, as the patient was only seen in life, uncertain.

† Brit. and For. Med. Rev., vol. xxiv.

‡ Barr, U. C. H., Males, vol. vii., p. 328-9. Here there was clicking pericarditic murmur at the second left cartilage and interspace; although the apex was raised, and the præcordial region bulged by fluid. Also Richmond, U. C. H., Females, vol. xv., p. 5.

121. *Extent.*—Pericardial murmur of one or other variety may be heard over all parts of the cardiac region from the roots of the large vessels to the apex. But I have never detected the churning variety except about the apex, nor the clicking variety except in the site of the larger vessels. The finest shades of grazing sound are most common behind the sternum. Different qualities of murmur may be heard at one and the same time in some cases over different parts of the heart; but this is not common. As a rule, friction-sound is most clearly and frequently detected below the third interspace, probably simply because, below that part, there is little lung intervening between the pericardium and surface. Limited in some cases to a mere point of surface, it may reach *from the clavicles to the epigastrium and from nipple to nipple.** In rare instances, friction-sound in the pericardium is audible in the back, between the scapulæ and the spine,—I mean in cases where mere intensity of sound cannot be held to explain the fact. It has been found single in the back, too, while double in front. It seems sufficiently likely that in some, at least, of the cases, where it is alleged to have been confined altogether to the back, the friction heard may have been produced in an inflamed pleura by the cardiac impulse. Pericardial friction is usually abruptly limited.

122. *Intensity.*—Pericardial murmurs vary exceedingly in intensity; so delicate in some instances, especially when of grazing or clicking quality, that the closest attention is required for their detection,—in other cases they may be heard in the posterior and lateral regions of the chest, and even, as already mentioned, at a considerable distance from the surface of the chest in the præcordial region [120, *a*]. On the whole their intensity is greater than that of pleural-friction sounds—a fact accounted for by the comparative abruptness and energy of the motion inducing them.

123. *Site of maximum Murmur.*—When the entire cardiac surface is the seat of friction-murmurs, the maximum amount of noise exists, according to some writers, about the nipple; to others, behind the sternum. I am satisfied no rule of the kind

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* Case of Mr. S., seen with J. H. Davis. The friction-murmur was in this case of the most unmistakable kind,—loud, grating, rubbing, jerking. This case alone would suffice to prove that friction-sound is not of necessity limited to the præcordial region.

can be established: I have known the point of maximum intensity change from the fourth interspace to the fifth, thence to the ensiform cartilage, and thence to the nipple, within twenty-four hours.[*] Neither can any particular part of the cardiac surface be fixed on as the absolute seat of loudest friction-sound; it is sometimes, though rarely, excessively loud even about the base.

124. *Synchronism or Rhythm.*—Pericardial friction may accompany both the systole and diastole, or either singly; its co-existence with the systole alone is not rare; with the diastole alone, infrequent. When of regular rhythm, the friction-murmur falls a little after the corresponding valvular sound. But such regularity as this is the exception, not the rule; the friction-murmur may be very distinct during the post-diastolic silence; and occasionally falls between the first and second heart-sounds. In point of fact, want of distinct synchronism of the murmur with either of the two sounds is a very habitual and distinctive attribute. Friction co-existent with the systole is generally, but by no means constantly, sharper and louder than with the diastole.

125. *Pitch.*—Pericardial murmurs are so deficient in tone, that their pitch can scarcely be estimated even rudely, although they embrace in their different varieties a considerable compass of sounds. And, indeed, no practical hint, that I know of, is derived from their pitch; except that, generally speaking, the higher this is, the drier and rougher the material of attrition. The pitch of a pericardial murmur may sometimes be raised by pressure with the solid stethoscope, and also by full inspiration.[†]

126. *Superficialness.*—Pericardial murmurs are, as a rule, distinguished by their superficial character: they appear to be produced immediately underneath the integuments.[‡] But if the physical cause of the murmur be placed beyond the limits of the triangular portion of the heart, uncovered by lung, they lose this character, and seem of more or less deep origin. This is sometimes particularly observable about the large vessels, before any effusion has occurred to push the edges of the lungs aside; their apparent depth then contributes to assimilate them in certain varieties to valvular murmurs.[§]

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[*] Campion, U. C. H., Females, vol. vi., p. 260. Here, at one time, it was loudest at the base.

[†] Bingley, U. C. H., Males, vol. vii., p. 114.

[‡] Calkin, U. C. H., Females, vol. x., p. 338, Nov., 1854.

[§] Skoda attempts to show that the clinical distinction of sounds, produced deeply and superficially, is a delusion. If a stethoscope be placed on the chest of a dead

127. *Simulated exocardial Murmur.*—If the præcordial interspaces be hollow from emaciation, the heart's action may produce a rustling sound by causing *friction of the skin against the stethoscope.* In a case of agglutinated pericardium, now under observation, this sound, well audible during expiration, when the surface of the intercostal plane rises against the end of the stethoscope, placed on the two contiguous ribs, ceases altogether while that plane sinks inwards during the succeeding inspiration.*

128. *Modifying circumstances.*—Various circumstances modify the intensity and superficial extent of these murmurs,—the physical condition of the pericardium itself remaining the same. The most important of these is the energy of the heart's action: the greater this, the louder the friction-sound. The weakness immediately ensuing on blood-letting, approaching syncope, the action of colchicum and aconite, all lower its intensity: if successive systoles be of very unequal force, friction, absent with one, is present with another, impulse. An hypertrophous and dilated heart gives, *cæteris paribus*, the maximum quantum of attrition-murmur. Attrition-murmurs are more marked in expiration than in inspiration; and when the trunk is bent forwards, than in the recumbent position. If there be fluid present, the friction-murmur may be less marked at the lower sternal region in the sitting than the recumbent position, doubtless from gravitation of the fluid.†

129. *Time required for production.*—Pericardial friction may appear very rapidly after the cause of inflammation has acted. Thus, in the remarkable case at University College Hospital where fatal perforation of the œsophagus and pericardium had been produced in the attempt to swallow a sword, friction was detected by my then Clinical Assistant, W. Tidmas, *thirty minutes after the accident.*

130. *Duration.*—The duration of friction-murmur varies very greatly. I have known it to appear and disappear finally (and in as much as the disappearance was final, it could not have depended on the occurrence of liquid effusion), within six hours—of the grazing variety, it is true, but still of unmistakable characters. In illustration of the other extreme, I may refer to a case in which

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body, the sounds produced by tapping different parts of the inner surface of the parietes, near and distant, will, as far as I have been able to ascertain, differ very sensibly; exceptions occur, it is true; but these do not destroy the rule.

* Aubrey, U. C. H., Males, vol. xvii., p. 268, Nov., 1860.

† Mison, U. C. H., Females, vol. xv., p. 176.

it continued audible for upwards of three months, especially at the lower part of the sternum.[*]

The total disappearance of pericardial murmur may, if it be slight in amount, almost immediately follow general or local bleeding; this disappearance may be only transitory, however, from weak action of the heart: or a pericardial murmur may abruptly disappear from rapid agglutination of the entire of the affected surfaces. I have known it impossible to find a vestige of friction in a case where, six hours before, the *entire* cardiac region was the seat of very intense rubbing sound. But, generally speaking, the progress of agglutination is materially slower than this; and for several days one or more limited spots may be found, where the collision continues soniferous (sometimes in the churning variety), after the adhering process has commenced. The sudden cessation of friction-sound, through absorption of exudation-matter, is less easily conceived; and where such cessation occurs, independently either of agglutination, or of rapid pouring out of effusion, the inflammation had probably produced very little indeed of that matter.

In the majority of cases where friction abruptly disappears, the change depends on fluid effusion, separating the pericardial surfaces from each other: it is consequently oftener an evil, than a good, sign. The amount of liquid required to annul friction-sound varies with collateral circumstances, such as the size of the heart, conformation of the chest, absence or presence of adjoining pleural adhesions, and the accident of partial soft adhesions having, or not having, occurred, before the fluid begins to accumulate. A case of Taylor's (loc. cit.) shows that friction may continue when eight ounces of fluid, or thereabouts, have accumulated: but generally less removes it. In a remarkable instance under my own observation friction-murmur was well heard a few hours before the death of an individual whose pericardial sac contained *sixty fluid-ounces* of liquid effusion.[†] As fluid accumulates, changes may sometimes be traced in the characters of the friction-sound,—increasing weakness and softness being their predominant character.

[*] Kennedy, U. C. H., Males, vol. i., p. 67. First heard on the 27th Oct., it was last heard on the 5th Feb. following, long after the man's discharge from the hospital, and apparent restoration to health. How much longer it may have continued I know not, as the man was not afterwards examined, though often seen again on duty in the streets as a policeman—a fact showing the completeness of his recovery.

[†] Bartlett, U. C. H., Males, vol. iv., p. 292.

Disappearing with the occurrence of effusion, friction-sound pretty frequently returns when the fluid is absorbed: this returning, or redux, friction generally, but not always, appears first about the great vessels and base. Pleuritic friction is very commonly caught at the redux period; pericardial, certainly, more generally at the outset, of the inflammation. The second disappearance of friction may be abrupt, slow and gradual, irregular with recurrences, or rapid over the heart generally; while in a single spot or two, most commonly either at the apex or about the great vessels, some form of the murmur remains for a time.

131. *Anatomical conditions.*—Friction in the pericardium, the cause of which lies within the sac itself, signifies inflammation of the membrane. It is exceedingly probable that mere dryness of the surfaces will suffice to produce the grazing variety. I know from observation that fine vascularity of a very small surface, without a particle of lymph, may produce faint rubbing noise [375, *b*]; * but exudation-matter is its common statical element. In the great majority of cases, exudation-matter forms on both serous surfaces. Taylor relates a case where, the cardiac surface alone being affected, ordinary friction was almost completely absent: still, however, it appears to have been audible, single and systolic, to a slight amount about the base. If exudation exist on the posterior aspect of the sac only, friction will commonly be inaudible. And, in those rare cases where the product of inflammation in the sac is essentially purulent or strumous, the collision of the surfaces may be totally noiseless.

A former attack of pericarditis will or will not prevent the development of friction-sound with a new attack, according to the state in which it has left the serous membrane and cavity of the sac. If it have left a state of perfect agglutination behind it, then, unquestionably, friction is impossible; if of loose adhesions, new lymph may be thrown out between these, and friction will be developed.† But, probably, old pericarditis will always have a tendency to limit the extent, and regulate the site, of new friction-sound.

Calcification of exudation-matter within the pericardium will certainly,[*] and tuberculous and carcinomatous disease may be conceived to, produce permanent friction-sound; but I have not observed this in the two latter diseases.[†] Whether fibrinous, cancerous, or other matter within, and in the walls of, the heart, may, by simply elevating the cardiac surface, produce friction-sound, independently of pericardial irritation, I do not know from experience; but the affirmative is improbable.

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* F. Parker, U. C. H., Males, vol. iv., p. 177, Dec. 1848. A rub may attend the impulse at the apex with knocking sound, independently of any pericardial irritation, as far as can be even suspected; *e. g.*, cases of Sus. Roberts and Bonsey, U. C. H., Oct., 1850.

† Craddock, U. C. H., Males, vol. vii., p. 336, admitted for his eighth attack of rheumatic fever: for five of these he had been treated in the hospital, and had pericardial friction on every fresh seizure.

132. *Friction-sound of cardiac rhythm produced in the inflamed pleura.*—The distinction of pericardial, from ordinary neighbouring pleural, friction, turns mainly upon its difference of rhythm. But sometimes cardiac action produces friction in an inflamed pleura adjoining, the pericardium being unaffected. And I am disposed to believe, but so far on the evidence of only one case, that if the pleural lymph have by its contraction puckered both the pleura, on which it lies, and an adjoining part of the pericardium adherent to itself, the occurrence of cardiac friction-sound of this anomalous mechanism will be materially facilitated.[‡] The distinction of this kind of friction, pleural in site, and cardiac in rhythm, is not always easy: though commonly limited to the confines of the cardiac region in front, it may be audible in the back on the left side;[§] its intensity is generally increased by the act of breathing,—the pleural surfaces, being then actually in motion of one rhythm, are more easily thrown into motion of another, than if at rest; it seems sometimes limited to the time of expiration.[§] The following circumstances argue, but by no means irresistibly, in favour of friction of cardiac rhythm being of pleural, and not pericardial, origin: the limitation of the sound to either edge, generally the left, of the cardiac region; fixity in one or more particular spots; cessation total, or, what is more common, occasional with certain beats of the heart, when the breath is held; and very marked unsteadiness in the intensity and quality of the friction-sound. Local dry pleurisy, close to both sides of the heart, and productive of friction both of pleural and of cardiac rhythm, is sometimes followed by dry pericarditis: the presence of the latter inflammation can scarcely be affirmed, until effusion takes place; for the dis-

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[*] Jones, U. C. H., Males, vol. xvii., p. 315.

[†] On the contrary, indeed, I have known very positive elevation of the pericardium by carcinomatous nodules unattended with the least friction-sound; but the serous surfaces were smooth. Case of R. Smith, Multiple Subcutaneous Cancers, "Med. Times," August, 1852.

[‡] Shears, U. C. H., Males, vol. xv., p. 87.

[§] Wilkinson, U. C. H., Males, vol. ix., p. 284. But true pericardial friction, as already mentioned [128], is commonly most marked in expiration: *e.g.*, W. Price, U. C. H., Males, vol. vii., p. 214.

tinction of the true pericardial friction-sound is under these circumstances most difficult.*

133. *Distinction of pericardial from endocardial murmurs.*—Pericardial murmur is, in the great majority of cases, easily distinguished from that of endocardial origin by the following characters and circumstances: its rubbing quality; its superficial impression [126]; its abrupt limitation and non-transmission in the course of endocardial murmur; its changeableness in precise seat and intensity from hour to hour; the increase, it undergoes, in sharpness and extent, when the patient bends forward;† its want of perfect synchronism with, or fixed relationship to, the heart's sounds; and its being sometimes accompanied by friction-fremitus, which can scarcely be confounded with valvular thrill. Pericardial murmurs are commonly rougher than *acute* endocardial murmurs. Hope, indeed, held that a diastolic *rough* sound is of necessity pericardial, endocardial diastolic murmurs never possessing this character: but in this he was certainly wrong; an aortic diastolic murmur may be very rough. On the other hand cases occur where it is next to, or absolutely, impossible to decide on the peri- or endo-cardial origin of a murmur. This difficulty is oftenest felt about the base and great vessels, and when the sound is of clicking character. Sounds really produced at the valves of the aorta may not be transmitted along this vessel,—as a consequence simply of their feebleness. Again, if both divisions, systolic and diastolic, of a double murmur be loudest at the same spot, that murmur is probably pericardial; for, generally at least, the divisions of a double valvular murmur attain their maximum intensity at points slightly different. It has been said, if either heart-sound be well distinguished, and in a *perfectly unchanged condition*, through a synchronous murmur, the fact argues for pericardial mechanism: I believe that there is some truth in this proposition, fairly interpreted; but degree of perfection of audibleness of a sound through a murmur is a matter so delicate, that it cannot be clinically trusted to. Full inspiration will sometimes sensibly raise the pitch of a pericardial murmur [125]; but to a less degree it may, by possibility, similarly affect an endocardial.

* Two cases of this kind (Hayes and F. Smith) once fell under my notice at the same time,—U. C. H., Ward 4, October, 1850. Vide Clin. Lect. "Lancet," loc. cit., p. 389, 1849.

† This is a distinction, however, to be by no means implicitly trusted to. I have known endocardial increased, and indubitable pericardial murmur decreased, by this change of attitude. The normal pericardial rub, which is so frequently excited by excessive action of a healthy heart, sometimes actually disappears, if the trunk be held forwards.

And, in truth, it must be confessed that precordial murmur may present characters so deficient in intrinsic distinctiveness, that the diagnosis of its pericardial or endocardial origin is almost wholly to be made from the course and mode of progress of the murmur, and upon the attendant conditions. According to some observers the distinction may be established in difficult cases by the different effects on the murmur produced by stethoscopic pressure—increase of intensity being the attribute of the pericardial class. The distinction, I am satisfied, cannot thus be made; and, as the present seems an appropriate place, the general subject of the influences of stethoscopic pressure on the characters of murmurs may here be briefly considered.

134. *Influence produced on murmurs of the pericardium, endocardium, or orifices of the great vessels by external or stethoscopic pressure.*—The following propositions embrace the results of my experience on this subject. (1.) Whatever be the effect of pressure in any particular instance, that effect is better marked with the solid than the hollow stethoscope. (2.) In the majority of cases an existing murmur, whether endocardial or pericardial, reaches the ear with greater intensity, if the stethoscope be firmly pressed against the chest, than if it be lightly applied. (3.) This increase of intensity is in part the result of the more perfect conduction effected by firm application of the conducting medium. (4.) But in some cases there is actual augmentation of mass of sound. (5.) This augmentation is more frequently observed in the case of pericardial friction, than of other murmurs; and in certain instances of superficial grazing friction, where the collision of the pericardial surfaces is slight and consequently the resulting noise slight, both collision and noise may be greatly increased by pressure. (6.) But *per contra* the very reverse effect will ensue, if it happen that pressure be made in a case, where the already existing attrition is precisely of the amount best calculated to produce noise; if the freedom of attrition in such a case be interfered with by approximating the rubbing surfaces too closely, the amount of noise will be lessened.* (7.) Pericardial rubbing-sound may in persons with flexible chests be often artificially produced by pressure, though the membrane,—free from exudation or morbid vascularity,—be in fact perfectly healthy. (8.) The pitch of an existing pericardial murmur is sometimes distinctly raised by pressure. (9.) The quality of a pericardial murmur may

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* Lower, U. C. H., Females, vol. xiv., p. 259. Scratching superficial pericardial friction wholly stopped by pressure.

be rendered more grating by the same process. (10.) That a true endocardial murmur may be reinforced by pressure has already been mentioned,[*] but it may occasionally be thus produced *de novo* (when inaudible by the ear applied directly to the surface) by very forcible pressure. (11.) I have not known such production of murmur within the heart synchronous with the diastole: the phenomenon, as far as I know, is always systolic. (12.) The most common seat is the base (therefore at the orifices of the great vessels), or at the second left space and cartilage in the direction of the pulmonary artery. I have observed artificial murmur also in the second right space. (13.) Thinness and flexibility of chest-wall are important conditions of the occurrence in all positions; but especially to the right of the sternum. (14.) The murmur at the left second space is of course excited in the pulmonary artery; that in the second right in the aorta; that at the base, probably, on account of its greater proximity to the surface, in the pulmonary artery. (15.) I believe that I have heard systolic murmur at the left apex, having the attributes of the mitral regurgitant variety, produced wholly by pressure. But I have no post-mortem evidence to provet he proposition; and it is difficult enough to understand how the two things should stand to each other in the relationship of cause and effect. Still very certainly a mitral murmur may even in the adult be notably increased by pressure.[†] (16.) In exciting endocardial murmurs by pressure on either side of the sternum, it is important to use a stethoscope of small diameter at its applied end, so that this may be partially placed between the ribs. I have never observed an artificial murmur to the *right* of the sternum except when an instrument of this kind was used. (17.) The pitch of an already existing endocardial murmur is commonly raised by pressure. (18.) The pitch of an endocardial murmur, wholly artificial, is commonly rather high; its quality blowing or whiffing.

It follows, then, with sufficient clearness from these propositions, that the mere fact of increase of intensity taking place by pressure will not distinguish with certainty endocardial and pericardial murmurs from each other. But if by pressure the apparent area of generation of a murmur be increased, and above all *its rhythm*

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[*] Lewis, U. C. H., Females, vol. ix., p. 320; Gay, U. C. H., Females, vol. ix., p. 316; in both cases systolic basic. This fact was, indeed, long ago pointed out by P. M. Latham (Clin. Lect., vol. i.), who cautions the observer against being deceived by such artificial murmurs.

[†] Beere, U. C. H., Females, vol. x., p. 90; Craddock, U. C. H., Males, vol. x., p. 83.

changed, if from single it be changed into double,[*] then I am disposed to believe the proof of pericardial origin is conclusive.

135. *Influence produced on murmurs by intrathoracic pressure.*—The influence of *external* pressure in modifying already existent, and in generating new, murmurs, is illustrated by the analogous effects of *internal* pressure of various kinds and mechanisms. The more important of such effects may be grouped in the following manner.

A. Pleuritic effusion displacing the heart sidewards and twisting more or less the great vessels at their roots, occasionally gives rise to systolic, and in infinitely rare instances even to diastolic, murmur. Of the latter a very unquestionable example, as I think, has been referred to in another place.[†]

B. Mediastinal tumour pressing on either the pulmonary artery or aorta, and an aneurismal sac of the aorta bearing against the pulmonary artery, may give rise to systolic blowing murmur.

C. Pericardial exudation-matter embracing the great vessels tightly at their roots, and constituting another variety of pressure, may engender systolic murmur in the aorta or pulmonary artery, or both. So, too, accumulation of fluid in the pericardial sac pressing on the great vessels may, according to Martin-Solon, exercise a like effect.

D. Certain enlargements of the abdominal organs, pushing the heart upwards, in some instances appear to engender systolic murmur in the great vessels. But no such result necessarily follows; and in certain reported instances of this alleged mechanism, the question is encumbered with the possible influence of spanæmia.

III.—ANOMALOUS EXTRA-CARDIAC SOUNDS PRODUCED BY THE HEART'S ACTION.

136. The action of the heart sometimes produces rhonchal noise in the adjacent lung,—cavernous, and coarsely or finely bubbling. The rhythm of the rhonchi, and their persistence during suspension of the breath, disclose their mechanism.

137. A double rasping friction-sound, so loud as to be audible at two inches distance from the chest, existed in Swett's remarkable case of enlarged granular liver, where the pericardium and valves were perfectly natural [63, *b*].

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[*] Pearse, U. C. H., Males, vol. xv., p. 60, June, 1858.
[†] Diseases of the Lungs, 4th Edit., p. 259.

IV.—RESPIRATORY SOUNDS AS AUDIBLE IN THE CARDIAC REGION.

138. In the natural state the respiration is clearly audible over the entire cardiac region, though somewhat enfeebled below its centre. Enlargement of the heart widens the superficial extent of this feebleness; pericardial effusion does so also. Now, in proportion to the percussion-dulness produced, liquid accumulation enfeebles the respiration-sounds much more than solid heart-enlargement,—to so sensible an amount, indeed, that the excess is clinically useful *as an aid* in distinguishing these two states of disease under difficult circumstances. The difference in solid and fluid conduction-power explains the clinical fact.

Pericardial adhesions, especially if attended with close union of the pericardium to the ribs, and agglutination of the adjoining pleural surfaces, will maintain the natural amount of audible respiration in the pericardial region, through the course of a subsequent attack of pericarditis with liquid effusion. Under these circumstances, persistence of respiration in front of the heart would, as I have elsewhere suggested, take rank as a sign of pericardial adhesions.*

V.—VOCAL RESONANCE IN THE CARDIAC REGION.

139. In the state of health, vocal resonance is weak, it may be actually null, in the præcordial region. In some cases of pericardial effusion, the resonance is not only morbidly loud on the confines of the liquid, but acquires an ægophonic quality. In the only instance in which I have actually observed this, the state of the lung adjoining the distended sac accounted for the increase of resonance,—the fluid simply gave this an ægophonic quality and *pro tanto* raised the pitch.†

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* Clin. Lect. "Lancet," loc. cit. p. 144, 1849.

† Harley, Consumption Hospital, Males, vol. i., p. 76.

§ II.—GREAT ARTERIES.

SECTION I.—INSPECTION.

140. The outline and movements of the large arterial trunks are not visible to the eye in the state of perfect health. In very emaciated persons the pulsation of the aorta in the epigastrium may be seen, however; and, if the arch of that vessel lies unusually high, and the individual be thin, slight impulsive motion may be visible above the sternal notch. The carotid pulses can scarcely be seen, if the neck be tolerably well provided with soft parts: the same statement applies to the smaller arteries. No visibly expansile character can be detected in any of these motions.

141. With these exceptions, notable visibleness of arterial pulsation, especially with onward locomotion of the artery, is an unnatural condition, depending on (*a*) general or local excitement of the circulation; (*b*) special disease of the heart; or (*c*) disease of the visible vessel itself.

(*a*) General excitement of the circulation, it is well known, produces visible throbbing action in the innominate artery and carotids, which passes away with that excitement. The vessels, however, simply pulsate, and undergo no onward locomotion. Visible pulsation of the epigastric aorta may be similarly induced. The arteries leading to an inflamed part often beat visibly.

(*b*) Aortic regurgitant disease, as was first shown by Corrigan, renders the pulsation of the superficial vessels visible: each vessel moves forward too, in the direction of its axis,—vermicularly, if its outline chance to be tortuous. The radial arteries at the wrist, the temporals, and the posterior tibials behind the malleoli, are the usual seats of the phenomenon; but in highly marked cases the carotid, brachial, axillary, femoral and external iliacs, the abdominal aorta, where the abdomen is flat and thin-walled, distinctly present it,—nay, it has been detected even in the central artery of the retina* [631].

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* Fitzgerald, Brit. Med. Journal, Dec. 23, 1871.

The extent to which this appearance may be trusted to, as evidence of aortic reflux, will be inquired into with the clinical history of that affection [631].

(*c*) The impulse of arteries locally diseased, either dynamically or statically, is much more effectually studied by the touch than by the sight.

SECTION II.—APPLICATION OF THE HAND.

A.—IN HEALTH.

142. The systole of the ventricles conveys to the entire extent of the arterial system an undulatory and somewhat expansile motion, easily felt in vessels, of a certain size, lying within reach of the fingers, and known as the arterial pulse or diastole. Each arterial diastole is followed by a contraction or systole. In vessels close to the heart the arterial diastole is perfectly synchronous with the ventricular systole, but falls more and more behind it, in point of time, the further the artery from the central organ.* With this qualification, the synchronism of the pulse and ventricular systole is perfect in health; the altered rhythm of the latter, produced by changes of posture, and all other physiological causes, is impressed on the former. In diseases of the heart, various perversions of this synchronism occur, and have been already described [103, *i*]. The systole of the arteries, synchronous with the ventricular diastole, is, or is not, pulseless.

143. The characters of the pulse are subject to wide variation in many particulars. The more important of these variations—all may acquire diagnostic significance, either as indicating some special systemic condition, or some distinct form of cardiac disease—may be grouped as follows :—

The characters of the pulse are to be distinguished (*a*) in each individual beat, and (*b*) in a series of beats.

(*a*). Each individual beat may be :

1. Quick or slow;
2. Short or long;
3. Soft or hard,—compressible or incompressible;.
4. Loose or tense;
5. Empty or full;
6. Narrow or broad,—small or large;
7. Single, double, treble, or multiple.
8. With or without special character,—vibratory, jerking, bounding, undulating, wiry, filiform.

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* I have somewhere seen the calculation, that the time elapsing between the shock of the heart and the radial diastole at the wrist does not exceed a fiftieth of a second. And yet this is an interval easily appreciable, after a little practice, with two fingers, one placed at the apex-beat, the other on the vessel.

(*b*) In a series of beats the movements may be:

9. Frequent or infrequent;
10. Equal or unequal in force;
11. Rhythmically regular or irregular.

The majority of these terms are easy of comprehension, and signify conditions really appreciable by the finger. And if we employ them less frequently than our forefathers, the disuse has arisen, not because the characters themselves are figments of the brain, but because we now possess more trustworthy clinical guides, which were unknown to past generations.

The characters themselves may be variously and capriciously combined, as will be fully seen in the description of individual diseases. As a single illustration, it may be mentioned that a pulse may be at one and the same time *quick* and *infrequent*—a seeming paradox which the student will easily understand by remembering that the *quickness* refers to the abrupt manner of each beat, the *infrequency* to the relatively rare repetition of the beat in a series.

The *single* and *multiple* pulse will by-and-by undergo full examination. Meanwhile, it may be stated the term *multiple* is designed for beats in each of which either a double stroke (pulsus bisferiens or dicrotous), or a triple stroke (pulsus trisferiens, or tricrotous), or several strokes (polycrotous), can be perceived.

Irregularity of rhythm may occur in the different forms, already described as affecting cardiac action [103, *i*].

All the terms enumerated are considered to refer to the blood-wave. But the vessel itself and its surroundings exercise an influence on the characters of the pulse, which it would be a grievous error to ignore. Thus, the inelastic rigidity of the atheromatous radial of an aged person will simulate hardness depending on left ventricular hypertrophy; a very thin arm will give relatively a broader or larger pulse than a fat one; while local œdema will, according to circumstances, intensify or weaken force of beat.

194. The pulsation of the thoracic aorta can be felt only in one spot in health,—above the sternal notch. If the finger be pressed downwards in that position, the patient's head being at the same time bent forwards, slight impulse is, in the majority of cases, detected: where the arch lies high, there will, of course, be very distinct movement—sufficiently marked, indeed, to suggest the notion of possible dilatation of the vessel. True aortic impulse is felt directly in the middle line; impulse inclined to the right side originates in the innominate artery. The pulsations of the ab-

dominal aorta may be felt, if pressure, proportional to the thickness of the parietes and intervening viscera, be made with the hand or stethoscope. The movement is simply one of elevation, without expansile action, and of variable force. Except in very thin persons, it is impossible to feel the beat of the vessel laterally.

B.—IN DISEASE.

195. In the various forms of dilatation of the arteries, the dilated portion pulsates with undue force, unless it be more or less completely filled with solidified fibrine. The character of the movement changes to *hammering*,—with force sufficient in some cases to shake the entire trunk and limbs. Besides, the diseased vessel becomes, under certain circumstances, the seat of a double impulse with each contraction of the left ventricle; the arterial systole conveys to the hand an appreciable shock. The diastolic pulsation is expansile, as well as heaving in character; but the distinction of the former quality of impulse is not always practically easy. Now, in the case of the thoracic aorta, there is no artifice by which obscure expansion may be rendered distinct to the touch: in that of the abdominal portion of the vessel, the hands may be sometimes slipped deeply on either side of the vessel, and a double sideward impulsion sometimes detected, which escapes discovery when the examination is made in front only. Still, it must be confessed, the pulsation of the healthy aorta, when pushed forwards by an enlarged vertebra or tumour, cannot always be distinguished by this plan from that of enlargement of the vessel itself [755, *b*].

196. *Arterial thrill.*—Arterial pulsation is in various forms of disease accompanied with thrill, occasionally more intense than that of valvular origin. Simple peripheric dilatation of a vessel, especially if it be roughly calcified, is a more efficient cause, than sacculating aneurism, of this phenomenon; and a spanæmic state of the blood contributes greatly to intensify it, when other conditions are favourable. Thrill may, in such a combination of circumstances, be felt two or three inches beyond the limits of the dilated vessel. Certain morbid crases of the blood, especially spanæmia, will suffice, independently of any textural disease, to produce arterial thrill, which, though slighter in amount, is generally more diffused, than that of organic origin.

Thrill may sometimes, through pressure on a given point of a vessel, be produced beyond, or on the distal side of, the point compressed,—being imperceptible at the spot which is actually impressed by the finger.[*]

SECTION III.—INSTRUMENTAL EXAMINATION OF THE ARTERIAL PULSE.

197. The mode of examining the force and other characters of the arterial diastolic shock and blood-wave by the application of a finger or fingers to the vessel is that which has prevailed in all countries, since the pulse was first known.

198. The difficulty of noting results with precision and of comparing observations, made at different times and by different people, has, however, from time to time, led to the invention of instruments (the idea seems to have occurred first to Galileo), designed to measure, or, in addition, to represent graphically, the force and certain other characters of the pulse.

199. The earliest of these instruments, which appears to have had any vogue, was the sphygmometer of Hérissant, invented in 1834,—a contrivance especially intended to measure force of beat, and by no means deficient in ingenuity; but error almost unfailingly arose from variations in the manner of applying the instrument (a membrane, to be laid directly on the vessel, gave support to a column of mercury in a graduated glass tube), and the invention was soon forgotten.

Next in the field appeared the kymographion of Ludwig (a modification of the hæmodynamometer of Poisseuille), followed by the sphygmograph of Vierordt. Both these instruments, though too defective for general adoption, did some service in the hands of their inventors. The stamp of general approval was reserved for the sphygmograph of Marey,[†] which, better than any preceding contrivance, wrote the pulse in legible characters, and, in fact, produced a sphygmogram. But even Marey's apparatus proved far from faultless, the difficulty of regulating the pressure exercised by the spring, placed on the vessel, being the major practical drawback to its use. Modifications have been proposed by Béhier,[†], by Longuet,[‡] by B. Sanderson ; B. Foster; and by Czermak, who was the first, as I believe, to utilize photography as an aid, in accurately fixing the pulse-trace. The instrument of Longuet, of very different form and structure, is certainly free from some of the defects of that of Marey; but its extreme delicacy and sensitiveness rather constitute objections than recommendations to its common use, and it is, besides, far from easy of management.

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[*] Allen, U. C. H., Males, vol. xi. p. 332. The finger laid on the radial feels no thrill ; but, pressure being made by one finger, another, applied to the vessel further from the heart, is distinctly affected by thrill,—the impediment caused by the pressure doubtless throwing the blood-current into ripples.

[†] Physiologie médicale de la Circulation du Sang, &c., 1863.

[‡] Bulletin de l'Acad. de Médecine, vol. 33 ; 1868.

[§] Idem. vol. 33.

200. For full descriptions of the instrument of Marey and its modifications, the student is referred to the Treatise of the inventor, and to *ex professo* essays on sphygmography.* It may be as well to observe, that the essential defect of Marey's construction lay in the manner in which the mainspring was riveted to the framework. It was so fixed that the pressure on the pulse was always considerable, while its amount could only be increased to a very small, and not accurately measurable, extent. Burdon Sanderson to some degree, but by no means completely, remedied this serious imperfection.

F. A. Mahomed has succeeded better. In his modified instrument the mainspring is suspended in position by a hinge-joint, so that the minimum pressure used equals only the actual weight of the spring; while, by two other contrivances, increase of pressure can be managed at will, and, above all, its amount accurately measured (in ounces troy). These improvements greatly increase the trustworthiness of the sphygmograph, and render, to a certain extent, impossible that vitiation of the principal characters of the best-defined pulse (say that of aortic reflux), which was liable to occur, from varying and unmeasured pressure, with the original instrument.

201. The peculiarities of each instrument in itself require to be specially understood before the manipulator can, as it were, thoroughly trust it,—the rapidity of movement, and more or less smoothly gliding play of the travelling slide,† the amount of friction of the point of the writing lever, &c., &c., must all be known by experience.

202. In applying the apparatus (according to common usage to the right radial), it is essential it neither press too tightly, nor hang too loosely, on the arm, and that the screws acting

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* Balthazar Foster, M.D. The Sphygmograph. 1866. Burdon Sanderson, M.D. Handbook of the Sphygmograph. Lond. 1867. The Sphymograph, By F. A. Mahomed, "Med. Times," January, &c., 1872.

† Two tracings given in the elaborate and conscientious work of Lorain (Le Pouls; ses variations et ses formes diverses, p. 116, Paris, 1870), and taken from the same radial artery within two minutes of each other, but with two sphygmographs, differing in rapidity of movement, differ so completely in characters, that it seems difficult to conceive they could have been furnished by one and the same vessel.

on the spring and the lever be neither too tight nor too loose: inattention to these matters of detail will more or less completely interfere with the reliability of tracings. It is essential, also, that the arm be so fixed, as not to cause any interference with the blood-current; and, for purposes of safe comparison of various pulses, the observer should accustom himself to placing his patients, as far as possible, in one uniform attitude.

Again, the method of fixing the instrument, adopted by Marey, does not efficiently provide against lateral or vertical change of position in respect of the radius; nor can it be fairly said that any of the alterations, made by various persons in this country and abroad with the view of obviating this defect (those of Mahomed* seem the most effective), have proved completely successful. Careful and practised manipulation, then, is everything: without it, two perfectly different states of disease, and readily distinguishable by correct tracings, may be made to furnish identical or quasi-identical sphygmograms. Nor is it to be forgotten that even the most skilful operator may, and does, encounter great difficulty in ensuring a number of successive tracings of the same pulse (especially if a healthy one), absolutely identical to the minutest point, though the whole series may be generically and in main attributes the same. The rarity with which identical sphygmograms are obtained from the right and left radial of the same individual is possibly as much an evidence of perfection as of imperfection in the instrument.

203. A sphygmogram, or pulse-trace, consists of a series of figures, which may be roughly called curves,—each one of the series corresponding to a single arterial beat, and in time representing (with a qualification to be by-and-by explained) the period from the commencement of any one, to that of the next succeeding, ventricular systole. In each of these curves there are three elements for study: the line of ascent, or up-stroke, the apex, and the line of descent, or down-stroke. Upon the variations in these three elements depends the signification of each tracing; their relationship will be easily gathered from the subjoined diagram.

204. The conditions to be noted in the *line of ascent* or *up-stroke* (*b*,*f*) are its degree of verticalness, its height, and the absence or

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* In Mahomed's instrument, the old wings, by which it was bound to the arm, have been removed; the "check spring" has been abolished; and a spring roller placed on each side of the groove, in which the slide travels, to prevent vibration and oscillation, and to insure a perfectly steady, gliding movement. But no amount of necessity for improvements in detail will lessen the homage due to the brilliant inventor of the original design.

presence of sub-curve in its length. This line coincides in the main with the course of the ventricular systole: the shorter that systole, and the more abrupt its onward propulsion of the blood, and the greater the ease with which the systemic blood flows onwards into the capillaries, the more vertical will this line prove. F. A. Mahomed, conceiving that it is caused by the shock received by the blood-column on the sudden bursting open of the aortic valves, which is transmitted almost instantaneously throughout the whole arterial system, and imparted to the arterial walls, proposes to call it *percussion-stroke.*

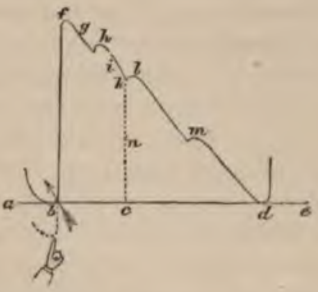

Diagram representing a single pulse-curve. a, b, c, d, e, base line, which, prolonged, unites the bases of a series of pulse-curves; b, f, line of ascent (or percussion-stroke of Mahomed); g, first portion of line of descent; h, tidal-wave; i, continuation of this, vessel collapsing; k, aortic notch; l, dicrotic wave; m, tricrotic wave; n, imaginary line, let fall from aortic notch perpendicularly on base line; point of index-finger, through b to c=ventricular systole; c to d=ventricular diastole; from point of index-finger to point at which arrow crosses base line and commencement of up-stroke= first cardio-arterial interval.

If the influx of blood into the aorta be slow, the line of ascent will be proportionally oblique; if that influx be uneven, the corresponding line will be irregular or sub-curved.

205. The *apex* corresponds in time to the moment of maximum dilatation of the artery, to the instant of commencing resistance to further impression by the blood-wave, and to that of commencing reaction of the latter on the vessel. The apex measures the time between the arrival of the blood at, and the onward passage of that blood from, a given point of the vessel. In the perfect order of things this line is too short to be graphically measured, and the apex is a mere mathematical point.

But while in the state of perfect health, local and systemic, the apex is a point or almost a point, in various morbid states it is prolonged into a line, of various lengths, horizontal in direction, or oblique upwards or downwards. Its prolongation indicates distal resistance to the onward current, or weakened elasticity of the vessel itself, by senile change or otherwise.

206. The *line of descent* or *down-stroke* commencing at *f*, the apex, is of much more complicated character than the up-stroke. It consists of the following subdivisions :—

Scarcely has the fall of the lever, signifying the collapse of the artery (*g*), commenced, when it is either simply stopped in its downfall, or (*h*) again slightly raised : this changed direction represents the true *tidal-wave*, according to F. A. Mahomed, which passes on (*i*) with a very slight curve,[*] concave downwards, till it is again arrested, as shown by the diagram, at the moment of the closure of the aortic valves, corresponding to a point (*k*), familiarly called the aortic notch. Here an upward movement again takes place, and another wave, the dicrotic wave (*l*), is produced. Henceforth the lever gradually descends, marking a simple oblique line, or yet another undulation takes place as shown at *l*,—a tricrotous wave.

207. These sub-curves (dicrotous and tricrotous), written and visible representations of the tactile impressions of double- or treble-striking pulse, already referred to [193], give new interest to the study, as old as the time of Galen, of doubling of the pulse. Long supposed to be a morbid phenomenon, Chelius was the first to show that dicrotism belonged to the healthy order of things.[†]

208. Uncertainty still hangs in some measure on the conditions favouring the occurrence of, or intensifying, dicrotism. Still it would appear that powerful systole of the ventricle, rapidity of the blood-current, notable capillary obstruction, great elasticity of the larger vessels, combined with slight amount of their diastolic dilatation and freedom of peristaltic action (F. A. Mahomed) in the arterial system, all more or less promote its occurrence. Burdon Sanderson appears to maintain that it mainly signifies low arterial, and high venous, pressure.

In the pathological order of things failure of muscular tone in the arteries, obstruction to the venous circulation, and tendency to stagnation on the right side of the heart, apparently intensify dicrotism.

Both in health and in disease the pulse may be tricrotous in vessels relatively near the heart, while dicrotous in those further off: respectively, for example, in the femoral and the dorsalis pedis.

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[*] The greater this curve, the greater is the difficulty of arterial collapse.

[†] O. J. B. Wolff maintains that the *normal* pulse of the radial and ulnar arteries is perceptibly *tricrotous to the finger* (Charakteristik des Arterienpulses, Leipzig, 1865, p. 142).

209. Numerous theories have been tendered, since the time of Galen, in explanation of dicrotism. Among the most plausible appears that ascribing it to the undulations produced by the mutual interference of the blood-waves, as they follow each other, transmitted to the vessel-walls. Vivenot shows that dicrotism occurs in artificial apparatuses, into which no imitation of the sygmoid valves has been introduced: this fact, as well as, better still, the occasional existence of dicrotism in cases of well-marked aortic reflux, seems fatal to the theory, which refers the secondary undulations to the tremulous influence conveyed by the fall of the recoiling blood-column on the sygmoid valves to the entire mass of the circulating fluid beyond. F. A. Mahomed maintains that dicrotism is essentially due to the contraction of the distended elastic walls of the aorta, which takes place immediately on the termination of the cardiac systole.

Tricrotism (diagram, *m*) is probably due either to recoil from the capillaries, or to secondary contraction of the elastic vessel-wall at the spot to overcome capillary resistance.

Dicrotism, while essentially belonging to the arterial systolic or collapse period, and to the line of descent, in certain rare instances synchronises with the period of arterial diastole, and is to be seen written in the line of ascent. In the former, the normal, case, Landois proposes to call the pulse *catacrotous;* in the latter, the abnormal, *anacrotous.* It is needless almost to add, that the mechanism of the secondary wave must wholly differ in the two cases [204].

210. *The relative duration of systole and diastole* would appear to be graphically demonstrable by letting fall a line (diagram *k*, *c*) from the lowest part of the aortic notch (*k*), in any given pulse-curve, perpendicularly on the horizontal line (*a*, *e*) uniting the bases of successive curves. But it has been acutely shown by A. H. Garrod* that a representation of the "first cardio-arterial interval" (diagram, from point of index-finger to arrow) must be added to the arterial interval (*b*, *f*, *k*) to give the true measure of systole, which will therefore stand from point of index-finger to *b* and thence to *c*. The "first cardio-arterial interval" represents the time between the commencement of the cardiac systole and that of the arterial diastolic line of ascent. The systolic and diastolic

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* Relative duration of the component parts of the radial Sphygmograph-trace in health. Proceedings Royal Society, May 19, 1870, p. 357, also 1871.

periods are, therefore, probably more nearly equal in length than in the ratio of $\frac{2}{5} : \frac{3}{5}$, which has been calculated from tracings of the normal pulse, without reference to the cardio-arterial interval. A. H. Garrod also shows (thus confirming clinical observations on the state of the heart's sounds) that in certain forms of disease, typhoid fever, for instance, the systolic period shortens.

211. So far we have spoken of a single pulse-curve. But the relationship of a *series of such curves* in an entire pulse-tracing requires consideration also. The points to be especially noticed are: first, the level of the apices of the curves; secondly, the level of the bases; thirdly, the number of curves in a tracing of given length (*vide* APPENDIX).

The line uniting the apices, which signifies the extreme amount of arterial tension, is level, because maximum tension remains a fixed quantity, in a perfectly normal pulse. In disease the line is, of course, liable to extreme irregularity.

The line uniting the bases signifies the greatest amount of arterial collapse in a series of beats: in the normal state (and if respiration be perfectly calm) this line is even and horizontal. If respiration be perfectly calm, we say, because the mere forcing of the expiration and inspiration-acts will, by increasing or by lessening pulmonary and systemic vascular tension, respectively raise or depress the lowest point of the pulse-curve, and so render the uniting line more or less uneven. Various conditions of disease, pulmonary and other, affecting respiration, consequently modify this line, which, to mark this significant attribute, has been called the *respiration-line.*

The number of curves on a tracing may be made to furnish the frequency of the pulse, if the length of the recording slide and the rate of movement be known. The finger applied to the wrist will, however, settle this point more quickly, as correctly, and much more readily.

212. Much has been written for and against the diagnostic utility of the sphygmograph. It has certainly disappointed the expectation of those who expected it to furnish a special tracing for every form of cardiac disease—in each case as significant as, for instance, diastolic left basic, or left apex, murmur. The instances in which a writing of the pulse will of itself decide the nature of a doubtful cardiac or arterial affection are certainly few in number. So far as it has yet revealed itself, the mission of the sphygmograph appears to be rather to indicate general conditions of the circulation (which may in the same form obtain in various and

unallied diseases) than to detect specific organic changes. The identification of such general conditions may be greatly more useful as aids to prognosis than to diagnosis.*

SECTION IV.—PERCUSSION.

A.—IN HEALTH.

213. In the natural state, the thoracic aorta has no appreciable influence on the percussion-sound of the surface, beneath which it lies. Even when the arch lies high, the resonance at the sternal notch, practically speaking, remains unaffected.

B.—IN DISEASE.

214. But when the vessel is notably dilated, dulness under percussion of course occurs, proportional in extent and intensity to the amount of enlargement,—modified in some degree by the fluid or solid state of the contents of the dilated part, and, to a serious degree, by the vicinity of the enlargement to the surface of the chest, and the condition of the intervening textures.

The dulness of a thoracic *sacculated* aneurism is not co-extensive with its dimensions. From its more or less globular form, a limited portion only of the sac reaches the chest-wall, and the observer cannot safely percuss with sufficient force to detect its deeply seated parts. Hence, practically, an intra-thoracic, and even an abdominal, aneurismal sac, is always larger than the results of percussion would indicate. In *non-saccular* dilatation of the aorta, percussion is, for obvious reasons, a surer guide to the size of the enlargement.

The resistance of a sac filled with fibrine is highly marked; and the deficiency of elasticity so peculiar, as to help in distinguishing the dulness under percussion from that of other states, for instance, from that of tuberculisation of adjoining lung. The line of union of simply condensed lung and a saccular dilatation is, however, with much difficulty to be drawn, even with the help of this peculiarity.

It is difficult to fix the smallest amount of dilatation of the arch of the aorta that can be clinically demonstrated. Much will depend on the pains taken in the investigation; much on the thinness of the chest-wall and the healthiness of other parts; but much more on the precise site of the dilatation. In a case where the dilatation increased the width of the vessel, when cut open, by two inches, and hence increased the diameter of the unopened vessel by only about two-thirds of an inch, the fact of enlargement, suspected from thrill and undue impulse, was *proved* by percussion. But here the right angle of the arch, the point where the vessel nears the surface most closely, was affected. So small an amount of dilatation could not have been detected in any other part of the arch.* Physical evidence will always be difficult to obtain, and sometimes prove wholly unattainable, where a small sac is situated at the posterior surface of the vessel. Still by careful percussion in the course of the arch, a sac as large as a good-sized walnut may be discovered, if it lie anywhere between the second right interspace and the left border of the sternum, and there be no special and unusual source of difficulty in the way, such as the fact of the aneurism passing directly backwards without widening the surface-diameter of the part concerned [751, *c*].

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* The influence of compressed air on sphygmographic curves has been elaborately studied by Vivenot, and the physiological teachings of the instrument been thereby both confirmed and increased in importance; but no new diagnostic hints have followed.

SECTION V.—AUSCULTATION.

A.—IN HEALTH.

215. *Arterial sounds.*—Two sounds, synchronising with the systole and diastole of the heart, are, in the state of health, clearly audible in the course of the pulmonary artery and arch of the aorta: gradually weakening in force in the thoracic division of the latter vessel, they are with difficulty caught in the lumbar region, but by depressing the abdomen with the stethoscope, may readily be heard in front. In some persons, however, a single sound, synchronous with the systole of the heart and diastole of the vessel, can alone be detected below the chest.

In the carotid and subclavian arteries almost always, in the axillary and femoral in a fair proportion of cases, the sound continues double; in vessels more remote from the heart, a single sound only, synchronous with the arterial diastole, can be heard. The more active the circulation, the larger the vessel, the thicker its walls, provided their elasticity be not impaired, the more marked is the sound perceived on auscultation. It is not unusual to find a single, very rarely a double, sound even in the brachial and radial arteries, where these conditions are favourable.

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* H. Gordelier, U. C. H., Males, vol. iii., p. 331, and Consumption Hospital, vol. i., p. 56: the patient was extremely thin, too; an accidental aid of no mean importance.

216. In the vicinity of the heart, the arterial sounds closely resemble the cardiac, in quality, pitch, and proportional duration; at a short distance from that organ, both become equalised in length and loudness; sometimes in the carotids the second is the longer and louder of the two. At any distance from the heart the sound loses tone completely.

If the vessels are full, and the blood of natural composition, the arterial sound is duller, but more prolonged, than when the mass of blood is small, and its quality thin. The arterial sounds are louder in females, children, and thin persons, than in males, adults, and stout people. Their intensity is increased by slight pressure; a blowing murmur takes their place, in a large proportion of persons, under stronger pressure.*

217. The arterial sounds heard in the vicinity of the heart are, doubtless, in great part transmitted from that organ: as the second can rarely be heard at any distance from the heart, the inference indeed arises that it is solely a transmission-sound, and that the systole of the arteries, at least of the minor ones, is noiseless. But that the impulsion and friction of the blood against the vessels, and vibrations of their walls, during their diastole, with the current-like motion given to their column of blood by the form of the vessels, generates sound, cannot be doubted; it is not conceivable that sound, audible in the popliteal or radial artery, is the mere result of conduction from the heart. Besides, it is not very uncommon to find the first sound in the carotid artery, oftener in the right than in the left, stronger than that at the aortic orifice.

B.—IN DISEASE.

218. The natural amount of arterial sound is capable of increase or diminution both in the aorta and pulmonary artery.

But the only modification of clinical significance, falling under this head, is reinforcement of the second sound in the pulmonary artery,—a state connected by Skoda directly with hypertrophy or excessive systolic action of the vessel itself, indirectly with mitral constriction and regurgitation, and with hypertrophy of the right ventricle. This matter has already been considered in another place [109], and will be again referred to with the history of the particular diseases.

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* But it is an error to suppose that any amount of pressure will produce murmurs in all persons and in all vessels: the contrary may be hourly verified at the bed-side. The smaller the artery the more easily is murmur generated.

219. *Arterial murmurs.*—The arteries, like the heart, become the seat of murmurs, either from organic change or independently of this. They may be arranged as follows :—

1. Organic.
2. Inorganic.
   - (*a*) Hæmic.
     - From altered composition.
     - From coagulation or deposition.
   - (*b*) Dynamic.

220. *Organic* murmur in the intra-thoracic arteries varies in intensity from a scarcely perceptible sound to one audible even at a slight distance from the surface ; is limited to a small portion, or, more rarely, extends over a considerable tract, of the vessel ; presents all the varieties of quality noted in cardiac murmurs ; varies in pitch from that of the word *awe* whispered in inspiration, to a high whistling note ; is of distinctly intermittent rhythm, single, either systolic or diastolic, or double ; and either short and abrupt, or prolonged and slow.

221. The organic conditions of arterial murmur are referrible to (1) change of form of the vessel; (2) to a modified condition of its internal surface; (3) to both these states combined; and (4) to communication between an artery and some portion of the venous system.

(1) Peripheric dilatation, or simple lateral sacculation of an artery (especially if abrupt and well marked), by altering the direction of the blood-current, generates murmur, even if the internal surface be perfectly smooth. Narrowing of a vessel produces a similar effect, by increasing the friction of the blood against the walls at the constricted point.* Murmur of both these kinds is always synchronous with the pulse, and not rough, unless the blood be spanæmic, and the inner walls deficient in smoothness.

(2) Mere atheroma, unless accumulated in unusually large quantity, does not produce murmur; infiltration with induration-matter, by puckering the inner surface irregularly, roughens the sound ; erosions of the lining membrane do so more effectually ;

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* Corrigan, however, argues with great ingenuity, that it is not at the constricted point of a vessel, but within the area enclosed by the loose walls beyond, that murmur is produced ; and the fact mentioned above [196, *note*] concerning thrill produced in a vessel beyond a point of compression would support his view.

and calcification of the vessel, if it cause irregular elevations of that membrane, is still more efficient. Murmur is also produced by particles of exudation-matter studding the surface of an artery recently inflamed. This variety of murmur is always synchronous with the pulse.

(3) In the varieties of aneurism with diseased coats, form and surface are changed so as to produce murmur, which may be systolic, diastolic, or both, and attended or not with sound also. The conditions regulating the time and accompaniments of this murmur, will be described under the head of Aneurism of the arch of the Aorta [780, *e*].

(4) Intra-thoracic varicose aneurism, in all its forms, is attended with murmur synchronous with the arterial diastole, sometimes prolonged through its systole.

222. A certain amount of force in the heart's action is essential to the generation of arterial murmur: increase of that force will convert a soft into a harsh quality instantaneously; again, a well-marked murmur will sometimes disappear under the influence of slight faintness, falling far short of actual syncope.

223. Murmurs heard in the thoracic aorta, single or double, are often merely conducted from the heart. But if a murmur, audible at any part of the arch, be of notably different pitch, of greater intensity, and of harsher quality than a synchronous murmur at the aortic base, it may be inferred that a cause of reinforcement exists in the vessel itself. The only source of fallacy would be the chance coexistence of badly conducting materials over the base of the heart, with excessively good ones over the arch of the aorta. The characters of an arterial murmur will sometimes guide the observer partially to a knowledge of its anatomical cause; but the actual determination of this will mainly turn on the state of other physical signs [100, *a*].

224. *Inorganic murmurs.* (*a*) *Hæmic.*—(1) *From altered composition.*—Inorganic arterial murmur of this description is commonly softly blowing, if the vessel be ausculted without pressure. If pressure be used, it rises in pitch, and becomes sharply whiffing or whipping, resembling the sound produced by a quick stroke of a riding-whip through the air. As a rule, it is intermittent, single, never continuous, synchronous with the diastole, and, though occasionally prolonged through part of, never solely synchronous with, the systole of the vessel, and affects the arterial system extensively, instead of being purely local, as the organic variety.

In rare instances this variety of murmur is distinctly double in vessels lying within the ready influence of pressure; I have known it so in the femoral artery, for instance,—here the portion corresponding to the systole of the vessel may exceed the diastolic in length.*

The clinical conditions of this form of inorganic arterial murmur are certain of those of cardiac murmur of the blood-class, especially spanæmia. It is said that plethora produces it;—a statement I have been unable to verify clinically. But of this more by-and-by.

225. Midway between the organic and inorganic varieties stands the murmur of an *artery, healthy in itself, but pressed upon by an adjacent tumor.* Arterial murmur of this mechanism is commonly synchronous with the pulse, that is, diastolic in rhythm, in regard of the vessel. But, just as may sometimes be observed in arterial murmur artificially produced by stethescopic pressure [631], so, in the case of tumor-pressure, the murmur may be double, synchronising both with systole and diastole,—and this in the case of a vessel beyond the reach of influence by the stethoscope. I have substantiated the fact, for instance, in the abdominal aorta, when ausculted in the lumbar vertebral groove:† its importance in regard of the diagnosis of aneurism is obvious.

226. (2) *From coagulation or deposition.*—Slight roughnesses on the inner surface of an artery, probably insufficient in themselves to produce such effect, lead indirectly to the generation of murmurs by causing local deposition of fibrine.

Coagulation occurring on a large scale must directly generate murmur. I believe that I have heard murmur of this mechanism, extending outwards on both sides of the sternum, and apparently following the course of the pulmonary artery, in a case where life was brought to a rapid close by thrombosis within that vessel.

227. (*b*) *Dynamic murmurs.*—That dynamic influence on the part of the arterial walls plays an important part in certain diastolic (that is, systolic as concerns the vessel's rhythm) aneurismal murmurs, cannot be doubted: the reaction of the sac on the distending current, sent on by the heart's systole, is sufficient to disturb the flow so as to produce murmur.

It is possible, too, that some such influence may be at work in the cases of double inorganic arterial murmur a moment since referred to.

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* Gall, U. C. H., Males, vol. xvii., p. 272. Here there was powerful systolic murmur at the aortic orifice, but none of diastolic rhythm.

† Gosling, U. C. H., Females, vol. v., p. 130.

§ III.—VENOUS SYSTEM.

SECTION I.—INSPECTION.

228. It is not intended here to refer to the signs of *local* diseases of the veins, such as thrombosis, varix, or phlebitis. Now, such local changes being excluded, it follows that considerable distension of any particular portion of the venous system indicates the existence of obstruction in the connected main trunk, or in the right side of the heart itself.

229.—*Jugular distension.*—The internal and external jugular trunks are, on this principle, the veins most frequently found enlarged,—the right more commonly than the left, when one side only is affected. This obstructive distension, uniform or varicose, even if increasing the size of the external jugular almost to that of the little finger, is unattended either with change in the integuments, hardness or cordiness of the vein, or tenderness under pressure. The common cardiac cause of this condition is tricuspid regurgitation; a more rare one, simple dilatation of the right cavities of the heart: beyond the heart it depends on pressure on the superior cava, or innominate veins by intra-thoracic tumor or aneurism. So that in the one case it comes of too free, in the other of obstructed, communication between the cervical veins and the right auricle. Another infinitely rare cause of the phenomenon is aneurismal varix within the thorax.

Excess of enlargement on one side cannot be trusted to as significant of local pressure, instead of cardiac disease, as the cause of enlargement. Dilatation of the heart and tricuspid regurgitation may be attended with excess of enlargement of the left, as well as of the right, external jugular; such excess is indeed common on the right side, rare on the left.*

Jugular distension is very sensibly affected by the patient's posture. The more closely the head and neck are placed on the same horizontal level as the heart, the greater the fulness—insomuch that a greatly widened vessel in the recumbent position on the back may, in the sitting attitude, cease to be of sufficient size to attract attention. As the patient turns from side to side, something of the same kind may occasionally be witnessed: thus, in a case where in dorsal decumbency the left external jugular was notably the fuller of the two, it became sensibly the emptier when the patient turned on the right side.[*]

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* Couch, U. C. H., Females, vol. vii., p. 344.

230. *Jugular pulsation.*—The external jugular vein, oftener the right than the left, is occasionally the seat of visible pulsation, especially at its lower part near the clavicle. Irregular in amount and in rhythm, though obviously connected in the main with the *ventricular systole,* jugular pulsation wants the distinctness of an arterial pulse, and is rather an unsteady intermittent tremulousness than a series of well-defined beats. The effect of *inspiration and expiration* on the blood-movement in these veins partly explains the irregular rhythm; which may also, in part, be referred with extreme probability to the influence of the *auricular systole.* The impulse producing the pulsation comes visibly from below; and when a portion of the vein is emptied by pressure from the clavicle in an upward direction, it re-fills immediately from beneath, while the pressure is sustained above.

The amounts of enlargement and of pulsation are not directly proportionate: oftentimes indeed the reverse. There is a point of extreme distension at which the vessel is perfectly motionless.

Lancisi, the original observer of this phenomenon, supposed that it was produced by eccentric hypertrophy of the right ventricle. Hope, holding to this view, explains the impulse by the "impetuous recoil of the tricuspid valve," which repels the blood about to pass into the ventricle with such force that its impulse is propagated back to the jugular veins. Many persons maintain that jugular pulsation only occurs where the tricuspid orifice is too much dilated to admit of closure by its valve, whence ensues regurgitation into the veins during the ventricular systole. Parkes teaches that, in addition to tricuspid insufficiency, rupture of the valves at the junction of the internal jugular and subclavian veins is a necessary condition of the phenomenon.

I know of no facts positively showing the necessity of such rupture of valves; the vessels may be sufficiently distended to render their valves incompetent, which is all that is required.[†] Besides, the valves vary in position, sometimes higher, sometimes lower; the number of their curtains is not always the same, nor their relationships to the current identical: there may be, as is most frequent, two cusps,—or three, or even one only. The valve may be congenitally wanting on one side of the neck, present on the other,—or they may be deficient on both sides.

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[*] Couch, U. C. H., Females, vol. vii., p. 345.

[†] Thos. Denham, U. C. H., Feb., 1851, Males, vol. vi., pp. 69, 77. Here notable pulsation, both of the jugular and innominate veins, had existed during life; the valve was ascertained to be perfect, but enlargement of the calibre of the veins had obviously rendered it incompetent.

H. Bamberger[*] recognizes two kinds of jugular pulsation, a simple "venous pulse," and "pulsation of the bulb": in the latter variety the valves, he asserts, are competent; in the former incompetent.

But, as concerns the tricuspid orifice? Unquestionably jugular pulsation is most frequently met with in cases of tricuspid insufficiency, though by no means in all of the class; while, as I have decidedly observed it, where the valve was not demonstrably incompetent, in cases of dilated and hypertrophous right ventricle, I cannot refuse to admit that this latter condition alone may produce it.[†] I believe, too, slight pulsation may occur in pure palpitation, disappearing with the cessation of the fit. If the ventricle be hypertrophous, and the valve insufficient, the pulsation reaches its maximum. It is to be remembered, too, that respiration affects jugular pulsation, emptying the vein in inspiration, distending it in expiration. The parts played severally by respiration and cardiac action, may be distinguished by causing the patient to suspend his breath for a moment.[‡]

Extreme jugular pulsation may exist without the least visible throbbing of any other vein.

231. The *right mammary veins* may be knotty and pulsatile; I have seen this in cases of insufficiency of the tricuspid valve, and of intra-thoracic tumor.

232. But veins, much more distant from the heart than these, —the veins, for instance, of the *dorsa of the hands and feet*,—may be the seat of pulsations either of cardiac or of respiratory rhythm, or of both combined. Sir W. Jenner has very kindly favoured me

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[*] Beobachtungen über den Venenpuls, Würtzburger Medicinische Zeitschrift, p. 233, 1863.

[†] It is too constantly assumed by observers, that where the tricuspid valve is insufficient to close the orifice at death, it has been so during life also. What proof have we that an instinctive constriction of the orifice does not, except in extreme cases, accommodate the width of the opening to the capabilities of the valve? And, in truth, as I have learned since this passage was written, certain experiments by Kürschner seem to show that the valve may accommodate itself to a very widened opening.

[‡] The jugular pulsation sometimes noticed in pneumonia may depend either on cardiac influence, or on that of respiration and the pulsation of the contiguous artery combined; the fluttering movement of the vein on the affected side sometimes attending pleuritic effusion is, as far as I have seen, of the latter mechanism,—and may totally disappear in the erect posture, though well marked in decumbency (I. Harrison, U. C. H., Females, vol. ix., pp. 329, 345).

with the particulars of three cases illustrating these various rhythms. When the rhythm is cardiac, pressure on one of the pulsatory veins on the back of the hand increases the strength and distinctness of the pulsations (which equal in number those of the radial pulse) to the distal side of the point pressed on, annuls them to its proximal side; the respiratory movements exercise no influence on the pulsations. When the rhythm is respiratory, the vein collapses in inspiration rapidly, swells in expiration slowly; and, when pressed on, its pulsation ceases to the distal, increases to the proximal, side of the point pressed on. One of these three cases exhibits the co-existence of the two sorts of pulsation (respiratory,—and cardiac, by *vis à tergo* through the capillaries, probably) in an infant aged eighteen months, cut off with pneumonia secondary to tubercles. The reason why respiration and cardiac action should exercise this influence on distant veins in some cases of disturbed thoracic action, and not in others, seemingly similar, is yet to be discovered.

233. In all cases of apparently intrinsic venous pulsation, the possible influence of *contiguous arteries* must be borne in mind.

234. Sudden diastolic collapse of the jugular veins has been put forward as a sign of pericardial adhesion [395].

SECTION II.—APPLICATION OF THE HAND.

235. Thickening of the walls of the jugular veins sometimes arises in cases of tricuspid regurgitation of long standing. If in such a case those vessels pulsate, it is very probable their diastole will be perceptible to the fingers, especially towards the bulb; but I do not remember ever to have actually observed this.

236. Visible pulsation is in some instances attended with soft thrill,—a minor degree of the arterial phenomenon of the same name. The most marked example is furnished by that excessively rare diseased state, aneurism of the aortic arch communicating with the vena cava.

SECTION III.—INSTRUMENTAL EXAMINATION OF THE VEINS.

237. The sphygmogram of a pulsating jugular has been shown by many writers, including the original observer of this fact, Bamberger (loc. cit. [230]), to be *anacrotous*, that is *dicrotous* in the *line of ascent:* so that the converse disturbance of current by secondary wave obtains in the veins and the arteries. In the

latter the dicrotism is systolic, in the former diastolic, in regard of the vessels concerned. The first portion of the line of ascent is short, oblique, appears to precede the ventricular systole, and to depend on auricular contraction ; the second portion, much longer and more vertical, is the expression of the ventricular systole.

But a jugular tracing often exhibits a dicrotous sub-wave in the *line of descent* also. Besides, several sphygmograms, printed by Bamberger, sufficiently show the total want of uniformity in the different curves both in height, in direction, in manner of dicrotism, in breadth, and in the lines of maximum tension and relaxation, and exhibit rise in expiration and fall in inspiration. But this varied unevenness is in itself characteristic, and evidently depends on the multiple influences playing, almost synchronously, on the blood-motion in the jugulars.

SECTION IV.—AUSCULTATION.

238. It was long believed that the blood-current in the veins is, in the normal state, non-soniferous. Observations have, however, gradually accumulated of late years showing conclusively that in a large proportion of persons possessing all the attributes of the ruddiest health, venous murmur may be detected,—occasionally even in veins where *no pressure with the stethoscope* could interfere with the flow of blood within the vessels examined. In order to avoid needless repetition, I defer to a future paragraph an epitome of facts bearing on this part of the subject [241, *a*].

239. *Characters of venous murmur.*—Murmur occurring in the veins possesses one invariable attribute, that of continuousness, uniform or remittent.

(*a*) *Quality.*—In point of quality, venous murmurs are referrible to four types : the blowing, the whistling, the humming, and the modulated. The blowing varieties may be as soft as the respiration-sounds in health, sharply blowing, loudly blowing, as the sound heard on applying a shell to the ear, or actually roaring. Or, the murmur may be cooing or whistling. To the humming type belong various murmurs resembling more or less closely the noise of a humming-top, the buzzing of a fly, the singing of a tea-kettle, &c. Lastly, venous murmurs are sometimes distinctly modulated, consisting of a series of separate tones, capable of musical notation, recurring at tolerably regular intervals, and accompanied by a low hum, which gives the continuous character to the whole.

(*b*) *General characters.*—Inclining rather to softness than roughness, and of moderate intensity, inaudible unless the ear or stethoscope be applied directly to the surface, generally of low pitch, as the whispered word *who*, (when modulated, of course, this is variable,) venous murmur is liable to change in intensity and quality from one moment to another. This change occasionally occurs from some intrinsic untraceable agency; more frequently from some one of the following causes. Acceleration of the circulation intensifies venous murmur; and as inspiration favours the rapid flow of blood in the veins adjoining the thorax, in these veins, at least, that act ought to, and does actually, increase the loudness of an existing murmur. But, on the other hand, suspension of the breath at first exercises even more markedly the same effect; the sharp collision of the blood disks *inter se*, and against the walls in the struggle to move onwards, and the increased vibration of the walls themselves, probably explains this. If the breath be held for any time, the murmur disappears. Any posture which stretches moderately the vein under examination, intensifies its murmur: if the part examined be a muscular one,—the thigh for example,—there is a source of fallacy in the rumbling sound of muscular contraction, which must be guarded against by auscultation in a perfectly relaxed condition of the muscles. In the neck, murmur is stronger in the erect than in the lying posture; doubtless from the greater rapidity of flow in the former attitude. Venous murmur attains its maximum under a certain amount of pressure, ascertainable in each instance only by actual experiment. Less or more pressure weakens and finally obliterates all audible sound. Sex exercises no influence on the intensity of venous murmur, nor, directly, on its frequency. No doubt, it is greatly more commonly observed clinically in females than in males; but this depends simply on the disproportionate frequency of its physical conditions in the two sexes. No evidence has ever been adduced showing that a given state, which fails to generate murmur in a male, will succeed in the case of a female.

(*c*) *Veins in which audible.*—The veins in which murmur occurs, may, as far as I have observed, be arranged as follows, in order of frequency. The *external and internal jugulars*, on both sides, or on one side only, in the latter case most frequently the right; the *subclavian* veins; the *femoral* (I have never failed to find it in these vessels when well developed in the neck, and it may be caught in them sometimes when inaudible in the jugulars); the *axillary*, the *superior cava* and *innominate* veins;* the *veins* *of the bend of the elbow*; certain *abdominal veins*;[†] the *pulmonary veins*; and the *superior longitudinal sinus*, especially at its termination in the torcular Herophili.[‡]

(*d*) *Rhythm.*—Invariably continuous and never intermittent in rhythm, murmur in a vein may be simply continuous, that is, of equable force constantly; or it may be remittently continuous, undergoing reinforcement and weakening at regular intervals. The type is humming when the rhythm is thus remittent. Two causes of this remittent character have been suggested: the pulsations of an adjoining artery against the soniferous vessel, which are supposed to give, at regular intervals, a momentary impetus to the current in the interior of the vein; and the co-existence of ordinary intermittent blowing murmur in the accompanying artery.[§] It seems unquestionable, the majority of instances are fairly explicable on one or other of these principles; some, which are not so, may, it would appear, be explained by intrinsic inequality of force of current in the veins,—an inequality which, we know, positively exists in cases of venous pulse.

(*e*) *Accompaniments.*—Venous murmur may be accompanied or not with arterial or cardiac murmur, the latter basic in seat and systolic in time.

240. *Site of continuous murmurs.*—But do the murmurs, just described under the name of venous, really deserve the title? The history of opinion on the point is exceedingly curious. Long after the ingenious and logical inquiry of Ogier Ward had demonstrated the venous site of the cervical "humming-top" murmur to the satisfaction of observers in this country, Bouillaud, and French writers generally, persisted in localising it in the carotid arteries. Skoda, treating of it in 1842 in his section on arterial murmurs, likewise placed it in the carotid, and actually ridiculed the idea of venous origin.[‖] And now that he has adopted the correct view,

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[*] M. Clarke, U. C. H., Females, vol. iv., p. 261, Oct. 1848, "Lancet," March 31, 1849.

[†] Sus. Roberts, U. C. H., Females, vol. v., pp. 218—220, Oct. 1850. Continuous loud "roaring" hum, coupled with arterial intermittent murmur, of maximum strength a little above and to the *left* of the umbilicus, but audible even to the right of the middle line. Here, too, appear cases of continuous hum, audible on deep pressure at the right edge of *some* enlarged spleens.

[‡] Davis, U. C. H., Females, vol. iv., p. 138. In this case a continuous remittent murmur was also audible at both sides of the mid-dorsal spine.

[§] I once heard, in a female child, a singing musical murmur at the right sterno-clavicular angle, apparently of a purely *intermittent* rhythm; but careful listening convinced me the murmur was really in the veins—that it was probably caused *statically* by pressure of the aortic arch against them, and *dynamically* given the seemingly pure intermittent rhythm by pulsation of that artery against them.

[‖] Percussion, &c., 2te Aufl. p. 199.

his countryman, Kiwisch, experimentalises, and writes, to prove that the veins are never the seat of murmur, the carotids being the real site of the cervical hum in a chlorotic woman. Kiwisch's argument, however, seems to me simply to present one more illustration of the unfortunate facility with which, in matters medical, plausible ingenuity may succeed in the effort to make "the worse appear the better reason." The following, among other facts, appear, in truth, conclusive as to the venous origin of these murmurs:

1. The gentlest pressure on the external jugular above the point on which the stethoscope rests, instantaneously silences the hum beneath,—an amount of pressure so slight as to be incapable of exercising the least influence on the neighbouring artery. 2. Continuous hum and either arterial sound or arterial intermittent blowing murmur may in some cases be heard at one and the same moment separately; I have repeatedly noticed this in the femoral artery and vein. 3. Continuous hum can be heard, where there is no artery to furnish murmur,—as, for instance, along the longitudinal sinus and at the torcular Herophili. 4. The erect posture increases, the lying posture lessens, continuous murmur in the neck; the precise converse effects are observed below the groin; this is only intelligible by the changes in the venous current produced by altered postures,—altered postures which exercise no appreciable influence on arterial action. 5. Continuous murmurs of different quality and different pitch can occasionally be heard over the external and internal jugulars; there is no difference of artery in the two situations.*

241. *Causation of venous murmurs.*—Various hypotheses suggest themselves in explanation of the occurrence of these murmurs, referring them severally to altered composition of the blood, diminution of the mass of the blood, change in the blood's motion, and change in the dynamic and physical conditions of the walls of the veins.

(*a.*) Venous murmurs are so constantly connected clinically with chlorosis and other affections, of which thinness of blood forms an element, that their generation in some way or other through that condition of blood was generally, and without, it must be confessed, actual proof, accepted as a fact. The deficiency of red disks in anæmia (hydræmia, or spanæmia) was held to explain the noisy movement of the blood,—a notion which received its first contingent of seemingly precise support from Andral, who, attempting to establish the exact relationship between the amount of spanæmic change and the constancy of venous murmur, found that if the red corpuscles fall below 80 per 1000, murmur is constant; if they range between 80 and 100, pretty frequent; if between 100 and 115, occasional; if between 115 and 126, very rare; if they reach the average of health, invariably absent. It is argued that the blood being unnaturally thin, the friction attending its movement is, in accordance with a law of Poisseuille's, increased to such an amount as to engender sound.[*]

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* Perhaps it is not altogether unworthy of notice, also, that Kiwisch failed to produce continuous murmur in the arteries of large-sized animals laid bare, and ausculted under various amounts of pressure.

But there are some facts obviously inconsistent with this theory, at least as an exclusive one. Thus it is well known that in cases of chlorosis, treated with iron, colour returns to the tissues long before venous murmur disappears; and, *vice versâ*, Becquerel and Rodier give analyses of the blood of two chlorotic girls, presenting well-marked venous hum, with a mean proportion of 125·1 per 1000 of red corpuscles,—certainly an amount falling within the limits of health. It was long since stated, too, by the "London Heart Committee," that murmur may be produced in the veins by pressure in a state of robust health; and I have heard it in women of florid complexion, who certainly were not at the time of examination (and, as far as I could ascertain, had never been) *symptomatically* anæmic.

Gradually observers grew prepared for the announcement, first, as I believe, made by Liman, that the venous current is soniferous, under the ordinary circumstances of stethoscopic examination, in a large proportion of children and adults totally free from appreciable disease. Many persons have subsequently gone over the same ground as Liman, and the following table, contributed by Winterich,[†] fairly represents the general results in *healthy* individuals.

PER-CENTAGE OF VENOUS MURMUR IN HEALTH.

Age.	Males.	Females.	Age.	Males.	Females.
1 — 5	97	98	30 — 40	80	86
5 — 10	94	95	40 — 50	77	78
10 — 15	89	95	50 — 60	72	75
15 — 20	86	88	60 — 70	68	71
20 — 25	82	88	70 — 80	40	39
25 — 30	80	86			

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[*] Rather "murmur" than "sound" in the auscultative sense of the word.

[†] Brit. & For. Med Chir. Rev., April, 1852. The inquiries of Herbert Davies may also be referred to.

From this table it follows, that normal venous murmur, almost constant in early infancy, gradually decreases in frequency with advancing years, attaining its minimum of about 40 per 100, in the decennial period 70 to 80. The average frequency of the murmur is slightly greater in females than males.

But, as far as my own observation goes, these returns do not wholly disprove the importance of spanæmia as an element of venous hum; for I have never yet succeeded in finding a strongly marked murmur of the kind in healthy people. The factors of the phenomenon in a minor degree exist in health; but in some way or other the clinical conditions of spanæmia are connected with its highly developed forms. There is a *normal* and an *abnormal* amount.

Is it possible that increase in the proportion of white corpuscles, a very positive attendant on chlorosis, and, as shown by Remak, on repeated hæmorrhage, plays a part in generating venous murmur,—such increase probably entailing increase of friction and labour in the circulation? It is certain that with disappearing anæmia and lessening hum the white corpuscles may be found to diminish;* but of course association does not prove causation. And I must confess my failure to discover any high degree of hum in any cases of leucohæmia (while it is certainly altogether wanting in some) would tend to invalidate this view.

(*b*) Pure diminution in the mass of the blood explains jugular hum according to Hamernjk, by the whirling and eddying motion into which the dwindled venous current is thrown in passing along a vessel, which in consequence of its anatomical arrangement always retains a certain width at its lower part. But it seems impossible to prove this alleged influence of diminished mass and differing widths of vessel to the exclusion of spanæmia, inasmuch as diminution of the blood's mass seems never to take place without a fall in the proportion of red corpuscles.

(*c*) The influence of eddying and interrupted motion, however, seems difficult to contest, in face of the manifest part played by regulated pressure with the stethoscope in eliciting the murmur.

(*d*) If the veins contain less blood than natural, their walls become looser,—the increased facility of vibration, thus arising, accounts for the murmur, in the opinion of some. Vernois, in a modified form of this theory, looks on the prominent folds, into which the walls of the veins are thrown, as the cause of murmur, through the impediments they offer to the flowing blood. But as the sinuses of the dura-mater, with their quasi-rigid walls, give murmur, the non-necessity of such obstructive action is obvious.

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* M. Pitt, U. C. H., Females, vol. viii. pp 21, 57, Nov. 1851.

(*e*) Experiments have been made on fluids traversing tubes by Weber, tending to show by inference that the vibrations of the walls of the vessels themselves, rather than of the contained fluid against those walls, cause the audible murmur.

(*f*) The notion referring the murmur to contraction of the platysma myoides is the feeblest that has yet been advanced. How comes it, if such be the mechanism of cervical hum, that sound is heard only over those parts of the platysma corresponding to the jugulars,—and that it may be caught in situations, where no muscular substance exists at all?

(*g*) The theory, ascribed to H. Duchek,* that a cervical hum is of valvular mechanism, arising "when the valves are half opened and put into vibration by a sufficient rush of blood," seems untenable on the plain grounds that jugular hum has been heard when those veins proved valveless, and true continuous murmur is heard in parts of the venous system [239, *c*], which never are provided with valves. Besides, the importance of valvular vibration, as an element of cardiac sound, which Duchek assumes as the basis of the venous theory, is anything but proven [94, ϵ].

(*h*) The real *mode of causation* of these murmurs is consequently obscure enough. It seems to me impossible to ignore the influence of the composition of the blood; *in highly marked spanæmia, neither flaccidity of veins, pressure on their surface, nor even velocity of current, is required for the generation of murmur*,—it exists in the sinuses of the dura-mater, especially where the arrangement of these canals is such as to promote abrupt collision of opposing currents meeting at a conflux, as at the torcular Herophili.

(*i*) It is held by some writers, that as murmurs may be heard in the veins of healthy-looking persons, they cannot be accepted as evidence of a morbid state of the blood. Such "normal" venous murmur, as already observed, is relatively very feeble in the adult. And, inasmuch as venous murmur cannot be found by any means in all adults, there is clearly something not perfectly natural in those who present it. Even in children it cannot invariably be caught; and what is healthy in a child may be morbid in an adult,—take the single instance of the different states of Peyer's glands in childhood and more advanced life. The composition of the blood differs in infancy and adult age; it acquires new characters, again, in old age, and these characters may very possibly render the fluid less apt to generate sound, whence the less frequency of venous murmur in old age.

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* The Doctor, Aug. 1, 1872.

(*k*) I would sum up on this matter, clinically, by saying, that a state of blood in which the venous current becomes slightly soniferous, is not incompatible with apparently perfect health; but that intense, readily audible and diffused venous murmur, is characteristic of those morbid conditions of the fluid called hydræmia, anæmia, and spanæmia.[*]

242. *Diagnosis.*—The diagnosis of venous murmurs turns essentially on their continuous character; and is excessively easy, except when accidental circumstances occur to render that character obscure. This happens sometimes about the base of the heart anteriorly, and between the scapulæ in the back. Murmurs in the pulmonary veins are partially masked by the cardiac sounds.[†] In addition to its peculiar quality, pitch, inconstancy, ready influence by change of posture, as guides to the venous origin of the murmur, its rhythm in respect of the heart's beat will *sometimes* aid in connecting it with the veins. Thus a diastolic murmur at the base (the signs of *organic* disease at the cardiac orifices being deficient) must be venous, according to my experience;—at least a hæmic cardiac murmur of that site and rhythm has never fallen under my notice.

243. *Arterio-venous murmurs.*—(*a*) The occurrence of murmur in cases of intra-thoracic varix has already been mentioned: on the same principle, if a lateral communication exist in any part of the body between a contiguous artery and vein, so as to permit the current from the former to enter the latter, a murmur of whizzing or whirring character, and essentially intermittent, results, mingled with a continuous sound, specially engendered in the vein.

(*b*) It is conceivable too, that a murmur of systolic rhythm only, and consequently intermittent, shall be produced within the superior cava, closely at the line of union of this vessel with the innominate veins, in cases of highly marked tricuspid regurgitation. But I am not sure that murmur of this mechanism has ever fallen under my observation.

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[*] The importance of this state of the fluid seems indeed to have received the final demonstration by the experiments of Weber, who has shown that murmur is much more readily produced in tubes if an admixture of blood and water, than if pure blood, be used as the moving fluid. (Ranking's Retrospect, vol. xxiv.)

[†] Commentary on case of Clarke, Lancet, March, 1849, p. 332.

214. Bamberger affirms that in the variety of venous pulsation, distinguished by him as "pulsation of the bulb" [230], a distinct systolic *sound*, of greatest intensity in the site of the valves, and indubitably to be looked on as a "valvular sound of the veins of the neck," may be caught without difficulty (loc. cit. p. 234). This is the only item of evidence I can adduce in support of the notion that *sound*, as distinguished from *murmur*, can be generated within a vein; for my own part, I have never heard anything of the kind. Plainly, some of the objections just made to A. Duchek's theory of venous hum [239, *g*] do not apply to the present statement; while, on the other hand, the *onus probandi*, as to the power of valve-vibration to produce sound at all under the attendant conditions, rests with Bamberger.

ADDENDUM.

AUTO-AUDIBLE MURMURS.

245. Although our knowledge on the subject be as yet limited, it may prove not without advantage to group together such facts as have been established concerning murmurs audible by the individual himself, in whose frame they occur. Such murmurs, which we may distinguish as *auto-audible*, may be venous, arterial, endo- or pericardial.

(*a*) *Venous.*—That murmur in the cervical and connected veins is more or less distressingly audible by some sufferers from anæmia, producing various forms of "singing noise" in the ears and head, is well known.

(*b*) *Arterial.*—So, too, whiffing intermittent murmurs in the carotid sometimes become audible, especially in recumbency and on the side on which the patient happens to lie. A remarkable case is referred to in the description of aneurism of the aorta, in which murmur, not only auto-audible, but distinctly perceived by the bystander at a certain distance from the body, was heard through a considerable portion of the arterial system [780, *e*].

(*c*) *Cardiac.*—Patients, the subjects either of organic or functional cardiac disease, will occasionally describe noises, referred by them to the heart, and in which the physician at once recognises the characters of murmurs. Under these circumstances I have actually known endocardial murmurs exist of two kinds: systolic at the base, and systolic at the left apex. In the former of these cases the explanation seems easy; the murmur transmitted along the aorta, reached the carotids. But in the latter case of mitral regurgitation, the explanation is not so readily forthcoming. In one instance of the kind, where there was neither carotid whiff nor jugular hum, but the mitral murmur was strongly reinforced by a flatulent stomach, I cannot refuse to believe that the patient actually heard that murmur, as she described its characters accurately, and, her heart never having been physically examined, until she was seen by myself, she could not, as it were, have learned her lesson from others.

A mitral reflux murmur, distinctly auto-audible, may cease to be so, without any change having occurred in the loudness, quality, or pitch of the murmur itself, or in associated conditions, to account for such cessation.

(*d*) *Pericardial.*—Lastly, I have lately seen a case in which pericardial high-pitched clicking sound was distinctly heard by the patient: the sound, audible through the stethoscope, so precisely agreed in its characters with the description given by the subject of the peculiar noise he heard, that no doubt could be entertained on the question. But I am unable to explain why murmur in the pericardium (it wholly disappeared both to observer and patient with the disappearance of acute symptoms) should have been auto-audible in this particular case.

The remarkable murmur described in another place [120, *a*, foot note] was occasionally heard by the subject of it herself.

There is probably something peculiar in the acoustic apparatus, where these various murmurs become auto-audible. At least, it is very certain, mere loudness will not ensure even a cervical murmur being heard by the subject of it. I have within the last few days met with a combination of the loudest arterial and venous murmurs in the thyro-jugular district, in a case of cardio-thyroid exophthalmos, that has ever fallen under my notice [723, *f*]; here the patient was wholly incognisant of the existence of any unusual sound.

PART II.

DISEASES OF THE HEART AND GREAT VESSELS.

DISEASES OF THE HEART.

246. Diseases of the heart, as of all other organs in the body, are divisible into two classes: in the one, as far as can be practically discovered, the dynamics of the organ alone are at fault; in the other, structural change is, more or less, apparent. Cardiac affections are hence dynamic and organic.

Section I.—Dynamic Diseases of the Heart.

247. The different perversions of the dynamics of the heart, which are known clinically, may exist in association with structural disease, as well as independently of this. The distinction of the two conditions, the associated and the unassociated, sometimes simple enough, often proves in actual practice far from easy.

248. A review of some of the more important of the alleged rules for distinguishing the two classes will at once show their clinical insignificance.

(*a*) The inconstancy of the symptoms of dynamic, and the constancy of those of organic ailment, are strongly dwelt on, for example; but all the subjective, and many of the objective symptoms may disappear temporarily in cases even of extensive organic disease. Such disappearance with ensuing recurrence may even take place several times.

(*b*) The existence of secondary changes, such as subcutaneous œdema, and congestion of the lung, commonly proves the cardiac affection to be organic: but not always; for spanæmia, added to nervous palpitation, may induce œdema.

(*c*) If exercise relieve a disturbed heart, its affection is pronounced to be dynamic only; if movement increase the suffering, organic. This proposition might lead to an incorrect impression; for, it is certain, if spanæmia coexist with perverted action, exercise may be unbearable.

(*d*) If, in the intervals of attacks of disturbed action, the force and rhythm of the pulse and heart are natural, those attacks are said of necessity to be functional: an error; for the most perfect tranquillity of the organ *may* exist, from time to time, though its texture is seriously unsound.

(*e*) It has been taught that inability to bear particular postures, more particularly that of decumbency on the left side, in cases of disordered action of the heart, shows that organic mischief exists. Often true, this proposition occasionally proves false: women with spanæmia, palpitating, though perfectly sound, heart and left intercostal neuralgia, can rarely endure the posture in question.

(*f*) It might be supposed that in the different amounts of local and thoracic distress, experienced in the two classes of disease, would be found a trustworthy guide to their distinction. But the truth is otherwise. For, singularly enough, the amount of local suffering entailed by disturbance wholly, or in the main, dynamic is often greater—is, in the mass of cases, greater—than that produced by actual organic disease. The patient with grave organic mischief, that may kill him at any moment, say, marked aortic regurgitation, may be so free from cardiac suffering, as to express petulant annoyance that his heart should be made the subject of examination: another individual, oppressed with disturbed rhythm and perverted innervation of his structurally healthy heart, dependent on flatulent distension of the stomach, refuses to be persuaded of his freedom from mortal cardiac mischief. As a rule, apprehension is not often excited in the subject of structural disease of the heart, until the secondary and distant evils produced by the local morbid changes, whatever they be, begin to make themselves felt.

249. The truth is, careful and repeated physical examination alone can, in a certain proportion of cases, justify a positive opinion on this question; and even this aid will occasionally fail.

(*a*) For, in the first place, the mere existence of certain abnormal physical signs in a palpitating heart does not warrant the assumption, that textural change exists. Thus a basic systolic murmur may be simply anæmic,—it may by possibility arise from mere perverted action of the sygmoid valves,—or depend simply on the unnatural force and abnormal direction with which the blood-current rushes against the orifices of the great vessels. Again, a systolic murmur at the left apex of the heart may be generated by irregular action of the papillary muscles. Further, extension of the normal area of cardiac dulness even considerably to the right may depend on merely temporary obstruction of the blood's movement through the heart, leading to distension of its right cavities, and be wholly independent of any real dilatation or hypertrophy.

(*b*) Conversely, in the second place, the total absence of physical signs does not prove the heart to be in a perfect state of organic soundness; there are slight amounts of change in the heart's substance, of which the perverted signs (for, doubtless, such really exist) are beyond the penetration of the present day. Nor must the student forget that numerous conditions of adjacent structures may throw cardiac physical signs into the shade, and prevent the detection, unless extreme care be exercised, of any positive disease. Thus emphysematous or even bronchitic distention of the lung, causing this organ to overlap the heart to an abnormal extent, will conceal increased area of percussion-dulness by an enlarged heart; and horizontal conduction of amphoric note from the stomach or colon [72] may gravely deceive as to the actual outline of the heart. And such instances might readily be multiplied.

(*c*) Assistance in cases of doubt may be obtained from diathetic peculiarities and co-existent diseased states. Functional disturbance of the heart is connected more or less constantly and markedly with the following conditions: *Perverted innervation*, as in cases of hysteria, spinal irritation, uterine and ovarian excitement, and various neuralgiæ, intercostal, dental, &c.; *altered condition of the blood*, as in hæmorrhage, anæmia, gout, chronic rheumatism, and chronic disease of the liver; *nervous exhaustion*, as from sexual excess, masturbation, or spermatorrhœa; *mechanical interference with the heart*, as when the stomach or intestines are distended with flatus; *certain poisonous influences*, as those of tobacco, green tea, and various diffusible stimulants. But, on the other hand, unfortunately for the purposes of diagnosis, none of these conditions are inconsistent with the presence of actual organic mischief.

250. Finally, there is this comfort in the practical aspects of

the question, just considered, that, although it cannot be denied, cases occasionally occur in which it proves impossible to affirm or deny the presence of organic change (more especially perhaps, of fatty metamorphosis in its early stages), the instances, in which a painstaking observer is wholly baffled, are really rare.

§ I.—ELEMENTARY DYNAMIC CHANGES.

251. Functional disorders of the heart, which fall within the pale of clinical detection, are worked out by, or composed of, various disturbances of the *elementary dynamic properties* of the heart, *innervation* and *contractility*. Hence it seems likely to tend to a better understanding of those functional disorders, if we commence by an analysis of the demonstrable or possible perversions of the elementary properties concerned. Clinically, however, this study is in its infancy; physiologically, even, our information is unsettled and incomplete. Hence, to a considerable extent, not a few of the statements, about to be laid before the reader, are to be accepted rather in the light of suggestion than of dogmatic assertion.

252. The altered conditions of innervation and muscular action require to be separately considered.

A.—PERVERTED INNERVATION OF THE HEART.

I.—CARDIAC HYPERÆSTHESIA.

253. Hyperæsthesia of the cardiac system of nerves may be supposed to induce neuralgia, to exalt the tactile sensibility of the organ, and to affect similarly its special sensibility. Let us examine these possible changes seriatim.

(*a*)—NEURALGIC HYPERÆSTHESIA.

254. Patients occasionally seek advice for the relief of pain, the chief seat of which appears to correspond to a limited spot about the left fourth intercostal space and fifth rib (sometimes a little higher than this), and slightly outside the vertical nipple-line. They maintain, with as much confidence as can reasonably be looked for concerning a problem so difficult as the precise localization of pain, that the suffering in question is situated at a certain, but not great, depth below the surface. Remaining limited to the spot indicated for a variable term, the pain may eventually extend to the upper part of the front chest, reach the

left shoulder, base of the neck and occiput, extend downwards to, and not beyond, the elbow, or reach the tips of the fingers. Various other modes of radiation of this pain may occur, which will be more fully examined under the head of ANGINA PECTORIS.

The amount of suffering ranges between a sensation of little more than uneasiness and one of the keenest anguish. In character the pain may be shooting, grinding, constrictive, or rending.

255. This pain is not a form of intercostal neuralgia ; the three characteristic points of tenderness in the course of the intercostal trunk, at the vertebral groove behind, close to the sternum in front, and midway between those spots, are wanting. Neither is it seated in the cutaneous nerves ; as a rule, there is no morbid sensibility of the skin in the spot,—far from this, a sense of relief is frequently experienced from gentle and regulated pressure. Nor has it any other than a merely accidental connection with hysteria ; it is not only rare in hysterical women, but in the female sex generally.

The same description, *mutatis mutandis*, will apply to a pain experienced occasionally behind, or a little to the right of, the lower part of the sternum in persons free from dyspepsia, pulmonary or hepatic disease, but obviously the subjects of obscure cardiac mischief.

256. There can be little doubt that the neuralgia described is really seated in the sensory filaments of the cardiac plexus. And this excited sensibility may exist in an isolated condition, no motor disturbance of the heart ensuing,—or it may be associated with, or prove the excitant of, disturbance of motor innervation also, leading either to increased or diminished action.

257. The act of bending forwards, especially in the sitting posture and if accompanied with effort, as that of drawing on a boot, is often (but with much greater frequency in elderly than in young people) followed by a peculiar pain referred by some of those, who experience it, to the heart. It is relieved more or less quickly by stretching out the chest wall and pressing on the surface. I am far from clear as to the nature of this pain ; but, I believe it to depend on twisting of the pericardial costal cartilages, which have lost the flexibility of youth. Once produced in the manner described, it is very readily brought on by a very slight amount of the original cause, and I know more than one person obliged to exercise the greatest caution in order to prevent its recurrence.

258. To return to the main subject of this section, neuralgia of the cardiac plexus. Singularly enough this affection is less often met with in persons suffering from obvious and well characterised structural disease of the heart, than where the evidence of such disease is either wholly wanting or obscurely defined. It constitutes in its extreme degrees an essential element of angina pectoris; and with the account of this disease its origin, mechanism, and treatment are considered.

(*b*)—TACTILE HYPERÆSTHESIA.

259. The heart does not appear to be normally endowed with any high degree of common tactile sensibility. I draw this inference from the positive and negative results of vivisections; and from the only case of wound of the heart in the human subject, it has occurred to me to witness.* Besides, were the organ tactilely sensitive to any notable degree, it seems at the least strongly probable that violent palpitation would, under all circumstances, be more or less seriously painful. Yet we know, as a clinical fact, that the most distressingly violent jogging motion of the heart may, and does, take place in some persons without the least feeling of genuine pain.

260. Now, whether, with or without structural change, the heart ever becomes the seat of morbid tactile hyperæsthesia, may be considered unproven. On the one hand, it is unusual, to say the least, in cases of endocarditis, to find pressure in the præcordial spaces painful, unless pericarditis be superadded. And the very positive tenderness under pressure, noticeable in many cases of the latter inflammation, must probably be referred to the serous membrane itself. On the other hand, patients with dilated heart sometimes shrink from pressure, especially if abrupt, on the corresponding part of the chest wall; and I have known similar sensitiveness in cases of diseased aortic valves. On the whole it seems reasonable enough to admit the probability of a condition in which the heart becomes morbidly sensitive to physical impressions, though the fact be hardly susceptible of, or at least has not yet received, satisfactory proof.

(*c*)—SPECIAL HYPERÆSTHESIA.

261. Without attempting to dispossess the Hallerian doctrine of inherent irritability, as a cause of muscular action, and con-

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* U. C. H., Males,—case published by Boswell Reid, now of Geelong.

ceding the motor influence of the nerve-ganglia of the heart and of their rhythmic centres, still we cannot reasonably doubt that the degree of sensitiveness of the nervous expansion on the endocardial surface to the stimulus of the blood, must play a part in regulating the activity and energy of the systolic movements. There must be a normal standard of that sensitiveness, just as there is in the case of the pulmonary nerves in regard of the stimulus of atmospheric air.

262. If, then, there exist such a normal standard, increase above, or decrease below, par are conceivable states of disturbed dynamism. Are they observable ones? I have no certain evidence pro or con. But it has appeared to me sufficiently likely, that some examples of palpitation, for which no one of the acknowledged causes can be discovered, may really be engendered through such special hyperæsthesia. Just as abnormally irritating blood is an admitted cause of undue excitement of the endocardial surface, and (through that excitement) of palpitation, the nerves being healthy,—so, conversely, blood of naturally stimulant quality may prove the indirect cause of over-action through contact with an over-sensitive rete of nerves.

II.—Cardiac Paræsthesiæ.

263. Anomalous sensations, or paræsthesiæ, of various kinds may be experienced within and about the area occupied by the heart. The usual difficulty is exhibited by patients in their attempt to describe the precise nature of these morbid sensations. But in some instances they seem to correspond more or less closely to the formication, heat or coldness, numbness, tingling, &c., well known in the limbs; sometimes special to the organ, they are spoken of as a sense of rolling, bubbling within, twisting of, or sudden falling downwards or backwards of, its mass.

264. There is an anatomical reason for connecting these sensations with the cardiac nerves; they occupy precisely the same sites as the pain of simple cardiac neuralgia, or that of fully developed angina. There is, besides, a physiological justification for connecting the two things; these paræsthesiæ occasionally produce leipothymia, or actual syncope, palpitation, fluttering action of the heart, or irregularity of pulse without palpitation. They are sometimes, even in the male, accompanied with globus and sense of choking.

265. Such anomalous sensations are often made light of, because they do not amount to actual pain. Yet they are pro-

bably quite as significant, as really acute suffering, of an unsatisfactory and more or less dangerous condition of cardiac function. I am led to apprehend from cases which have fallen under my observation in private practice (they are rarely to be seen in hospitals), that where they unmistakeably exist, these paræsthesiæ indicate a want of perfect textural soundness in the heart. Nothing may be physically wrong,—auscultation gives apparently normal results: yet at any moment the mechanics of the organ may be dangerously perverted.

266. Cardiac paræsthesia, sometimes connected with passing congestion either of the cavities of the heart, or possibly of the actual tissue of their walls, is occasionally linked to organic disease, either of the muscular substance, or more commonly of the valves. But in the purely neurotic form it seems a reflex neurosis, in which irritation of the gastric branches of the pneumogastric form often the first link in the chain; even here, as just suggested, the probability is, some unhealthy condition of texture, too delicate for clinical discovery, affects the organ—otherwise it seems difficult to understand why cardiac paræsthesiæ should not be materially more frequent even than they actually are.

Agents which seriously affect the systolic contractile force of the ventricles, either by increasing its intensity (as, under certain circumstances, digitalis), or by weakening it (as aconite), produce, when exhibited in poisonous doses, various cardiac paræsthesiæ, sometimes actually acute pain.

III.—Cardiac Anæsthesia.

267. Of anæsthesia, or sensory paralysis, of the heart, little is known. Nothing has, indeed, as far as I am aware, been clinically ascertained concerning annulled tactile sensibility of the organ. On the other hand, impairment of its specific sensory innervation is so mixed up, nay, identified, with certain forms of failure of its motor power, that the subject may be most conveniently taken up in association with that of cardiac motor paralysis and syncope.

B.—Morbid Motor States of the Heart.

I.—Cardiac Motor Paralysis.

(a)—FROM PERVERTED INNERVATION.

268. Motor paralysis of the heart, dependent on perversion of innervation, is produced in four essential ways.

269. (*a*) It may be *centric:* as where intense emotion, either painful or pleasurable, a feeling of intense disgust, or sudden and general injury of, or shock to, the encephalon or cord, causes instantaneous cessation of the heart's contractions.[*]

(*b*) It may be *reflex through the ganglionic system;* as where a blow on the epigastrium causes fatal syncope.[†] It may be reflex, also, through the spinal nerves, as where severe injury to an extremity, or to an extensive islet of skin, produces fatal depression of the heart's action.

(*c*) Cardiac paralysis may also be induced through *direct interference with the supplying portions of the ganglionic system.* At least, cases now and then occur in which the pressure of enlarged bronchial glands, mediastinal tumor, or aortic sacculated aneurism on the cardiac nerves, has caused occasional interruption of contraction. In a very strange case of partial suspension of action for five or six weeks at a time, which ended fatally, the explanation was found in a knotty, diseased, and compressed state of the great cardiac, with the vagus, and the phrenic, nerves.[‡]

(*d*) The continuance of cardiac action after cautious and gradual piecemeal removal of the brain and cord, and the vitality of monstrous fœtuses, born without either of those parts of the nervous system, point to the non-dependence of the heart's activity upon them, and to the reality of intrinsic production of nervous force as the main source of movement. Remak's discovery of ganglia on the nervous filaments within the texture of the heart, seems to give the impress of positiveness to this view concerning the heart's motility.

Now, it is infinitely probable, *changes occurring in the ganglia themselves, either dynamic or structural*, may be the immediate causes of suspended pulsation of the heart in some instances; but the clinical proof is yet wanting. Still, in certain fatal cases of chloroform-inhalation, where death occurs through the heart (contraction ceasing sooner in the ventricles than in the auricles), it seems reasonable to believe the vapour acting directly on the cardiac ganglia, plays a part in instantaneously arresting their activity.

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[*] That the heart continues to beat after the *gradual removal* of the brain and spinal cord in animals does not shake the truth of this statement of facts, practically well known. Besides, it is certain that if the brain or cord be *suddenly crushed* in an animal, the heart ceases to beat.

[†] Marshall Hall proved that sudden stoppage of the heart's action might be caused by "violently crushing the stomach with a hammer," even where the communication through the cord and encephalon was impossible—those centres having been previously removed or destroyed (in an eel). Dis. of the Nervous Syst., 1841, p. 128.

[‡] Heine, Müller's Archiv, 1841,—quoted by Romberg, Dis. of Nerv. Syst., vol. ii.

The death, in a state of ventricular diastole, which results from poisoning by aconite, is also probably referrible to paralysis of the intra-cardiac ganglia.

(*e*) *Over-stimulation of the vagus nerve*, by greatly increasing the inhibiting force of that trunk [327, *c*], may lead, especially in certain disturbed states of the heart, to suspension of contractile function, the ventricles remaining in a state of forced diastole.

270. The practical relationships of this subject are considered under the head of SYNCOPE.

(*b*)—FROM EXHAUSTED CONTRACTILITY.

271. That deficient action of the heart depends sometimes on purely dynamic failure of the specific vital property of its sarcous element, contractility, the nervous supply remaining unchanged in quality and amount, seems hardly susceptible of proof. To demonstrate in any case of failing contraction, that the stimulus is positively unaltered, but the faculty of responding to that stimulus destroyed, exceeds the clinical power of the present day.

Nevertheless, analogy suggests the possibility of such a state. The sudden cessation of contractile action of the bladder in cases of stricture of the urethra, where immense force having been used to effect the discharge of perhaps not a quarter of its contents, any further effectual strain of its walls becomes impossible, most probably depends on temporary loss of contractility. The sufferer is at least conscious of vainly directing the full force of volition to excite the viscus to further contraction.

Besides, it would seem to follow from the experiments of Blake, that certain agents injected into the blood really do arrest cardiac action, not by perverting its innervation, but by annulling its muscular irritability.

272. No reference is of course intended here to cases in which statical change affects the sarcous structure. The minutest amount of such change confessedly and certainly nullifies in the most complete manner any amount of activity of motor innervation in the spots actually diseased.

II.—CARDIAC SPASM.

273. Does the heart ever become the subject of true spasm, either clonic or tonic? A positive answer, founded on clinical evidence, cannot in all probability be given to this question.

274. But Ed. Weber has shown that portions of the heart may be brought into a state of persistent tonic contraction, and cut off from the rhythmical movements of the rest of the organ, by the interrupted galvanic current and by the direct application of strychnia to the endocardial surface. Hence there is a possibility, at the least, that perverted motor innervation may cause more or less enduring spasmodic contraction, local or general, of the heart.

And is not the systolic contraction which destroys life in the frog poisoned with digitalis, a tonic spasm of the ventricular wall? Curiously enough, the same limitation of contraction to certain portions of that wall, noticed in strychnial poisoning, has been observed by different experimentalists (Dybrowsky, Pellikan, Fothergill), where digitalis has been the agent used.

275. Besides, it is not very uncommon to find the left ventricle so tightly contracted, on post-mortem examination of persons dying from various diseases, that it requires an effort with the fingers to open out the mitral orifice. I must confess ignorance as to the precise conditions preceding death in such cases; but the observations of Cruveilhier on simulated concentric hypertrophy of the heart throw some light on the matter. It is certain, at the least, that, in cases of death from tetanus, knotty spasmodic contraction of the organ has sometimes been observed. Some further notice of this subject will be found with the description of Angina Pectoris.

276. In all these cases over-stimulation of the cardiac ganglia would represent the nervous element at fault.

III.—Cardiac Cramp.

277. Cramp of voluntary muscle clinically consists of local tonic contractions, *plus* more or less agonising pain. It is commonly believed the pain specially depends on dislocation or rupture of muscular fibres, or possibly even of nervous filaments. Protrusion of the primitive fasciculi through ruptures in their containing sheath has been microscopically proved to occur in the voluntary muscles in cases of tetanus; but I am not aware that structural injury to nerve has been similarly demonstrated.

278. Such mischief in the sarcous structure of the heart remains, if real, to be detected. The phenomena of certain fatal anginal seizures make it, however, sufficiently probable that the search would not always be vain. Patients occasionally, and of their

own accord, liken the pain of angina to that of external muscular cramp.

IV.—Perversion of Rhythm and of Force of Cardiac Contraction.

(*a*)—OF THE ORGAN AS A WHOLE.

279. There are numerous affections of the heart of a statical kind which produce irregularity in the time and force of systolic action,—such as sudden rupture of a chorda tendinea, or of the substance of a valve, accumulation of coagula in the interior of the cavities, &c.; the irregularity in such instances becomes more or less readily intelligible on mechanical principles. But in a very large proportion of cases of irregular rhythm, there is no evidence of mechanical defect in the organ; we are obliged to admit in such cases either that structural mischief, beyond existing powers of clinical detection, exists, or that the disturbed rhythm is purely dynamic. The latter notion is in general more probably the correct one,—and to this form of perversion alone we propose now to direct attention.

280. It may be assumed that modified nervous influence is the intimate cause of dynamic abnormal rhythm. And that the modification may be (*a*) *centric* or (*b*) *reflex* seems amply shown by the clinical conditions under which irregular action is observed. Nor can there be any doubt, if we trust the results of recent physiological inquiries, that (*c*) *the intrinsic ganglionic system of the heart* may sometimes be independently at fault; though of this clinical demonstration is neither at present forthcoming nor likely soon to appear.[*] Thus, Volkmann's success in locally disturbing the rhythm of the organ by cutting and chopping its texture in various spots and manners, while it seems to show pretty conclusively that rhythmical action is under the immediate influence of local powers, limited in each instance to certain fixed areas, affords no clue to the comprehension of irregularity of beat occurring in the human subject. Nay, more, though we accept the existence of "rhythmic nerve-centres" as positively demonstrated, and admit that these centres in the nerve-ganglia, by discharge of nerve-force at regular intervals, induce the fixed

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[*] Still, when the most patient investigation fails to disclose anything at fault in the encephalon, medulla-oblongata, or organs connected with the systemic, or vagus nerves, it seems reasonable to conclude that the intra-cardiac nerve-apparatus lies at the root of the disturbance.

rhythmical contractions of the organ (mere motor power depending on quite a different agency[*]), we shall have long to wait before clinical demonstration of the part played directly and exclusively by mishap, dynamic or textural, to these rhythmic centres, can become a possible achievement. The problem of irregular rhythm is, in truth, one of infinite complexity. Thus, the readiness with which time-regulated action is perverted, is shown by an experiment of E. H. Weber, proving that even slight changes of temperature gravely modify the frequency of the heart's beat. How many other conditions must there not be, of like or greater potency, the influence of which the observer of the present day scarce even suspects ![†]

The reflex influence of the gastric branches of the pneumogastric seems clearly established clinically. The passing irregularity of action, which follows in some persons the mere ingestion of a full meal, and the graver and more continued disturbance, occasionally attending different forms of dyspepsia, seem otherwise inexplicable. And, again, the perversion of rhythm immediately induced in some exceptional persons by certain articles of diet, as green tea, is probably a reflex effect of specific influence on the vagus nerve, whereby the *co-ordinating* function of that nerve is temporarily annulled. Nor is the agency of the pulmonary branches less certain. A remarkable illustration, occurring in his own person, has been recorded by Nooth: a shot had lodged in a bronchial tube; harassed by asthmatic seizures and extremely irregular pulse, whilst the foreign body remained in its place, the patient lost both symptoms on its expectoration.[‡]

281. The different varieties of irregular beat have already been mentioned [58, *b*; 103, *i*]. A simple intermittence appears to

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[*] Pathology long since proved this by exhibiting force changed, while rhythm remained natural; or, *vice versâ*, irregularity of beat with power of contraction sustained at par.

[†] Many years ago Neil Arnott, in his "Elements of Physics," suggested that the rhythmical movements of the heart might be referred to accumulations of force, discharged at regular intervals, likening those movements to "some electrical phenomena in which there are successive accumulations and exhaustions of power." ("Elements of Physics," Fifth Edition, 1833, vol i., p. 597.)

Fresh interest has been recently given to the subject of rhythmic action by the lucid exposition of Paget, who, in a series of propositions of increasingly wide generalisation, finally endeavours to establish time-regulated rhythmic motion as not only a dependence upon, but a necessity of, *rhythmic nutrition*. (Croonian Lecture, Royal Society, May, 1857. I would strongly recommend the reader who may feel interest in the subject of cardiac rhythmical action, to study a very striking chapter in Herbert Spencer's "First Principles" (chap. xi., p. 313), embracing the general philosophy of the rhythm of motion.

[‡] Trans. Soc. Impr. Med. Knowledge, vol. iii., p. 5.

signify *pro tempore* cessation of power to contract, and may consequently be looked on as *a perpetually recurring, short-lived syncope:* in support of this view stands the fact, that some persons, subject to intermittent action, are conscious of instantaneously transitory faint feelings at the moment of its occurrence.

282. Although, as a rule, irregularity of cardiac rhythm must be looked on *per se* as of unfavourable import, it is indubitable that persons are occasionally encountered whose health is excellent, though their heart's action is more or less constantly irregular. The constancy of irregularity is subject to great variety,—inexplicably so for the most part. Individuals whose heart's beat is habitually irregular in ordinary health may have perfectly rhythmical action during pyrexia, either as a constant or an occasional state, so long as the febrile excitement endures. Subjective consciousness of irregularity, either in the form just referred to, or in any other shape, is by no means invariable, and is more common where palpitation is present than where this is absent. There are certain idiosyncrasies mixed up with this perversion of very singular character: the case referred to above, where a very small quantity of green tea will, in persons whose ordinary beat is rhythmical, immediately induce irregularity, is a sufficient illustration.

Another strange fact is, that sufferers from so-called cardiac dyspepsia and distress may feel much more comfortable, when their rhythm is irregular, than when it becomes (as it occasionally will) perfectly regular.

(*b*)—OF DIFFERENT PORTIONS OF THE HEART IN RESPECT OF EACH OTHER.

283. The natural synchronism of action in the two ventricles, in the aorta and pulmonary artery, and probably, though less demonstrably, in the auricles, may be variously perverted.

Of these perversions of action, which are solely revealed by auscultation, the history, as far as I have made it out, has been given under the head of Reduplication of the Heart's Sounds [p. 73].

284. The different conditions of action in different parts of the same ventricle, seen to occur at one and the same moment in the frog poisoned by certain agents,—one part being in systole, others in diastole simultaneously [p. 173]—show that there is a sort of independent action of the nerve-centres, though this independent

action may be, and is, subordinate to a general controlling power in the healthy state. On this principle, irregularity of action of the papillary muscles (with its effect, dynamic reflux [p. 89]), the heart as a whole acting regularly, becomes easily intelligible. And, as affording a further clue to the comprehension of clinical lapses of the present class, may be recalled the fact that power of rhythmic contraction may be retained by every small fragment of the border region of the auricles and ventricles (a sort of auriculo-ventricular belt), after all power of contraction, except that of a single response to a fresh extrinsic stimulus, has been lost by the rest of the heart. The experiments of Paget on the heart of the tortoise after removal from the body demonstrate this.

V.—Action Perverted in Frequency.

(*a*)—INFREQUENT ACTION.

285. It is conceivable the heart shall contract less frequently than natural, either because (1) normal innervation is in some way perverted; or because (2) nervous influence continuing normal, the contractility of the heart's fibre and its faculty of responding to stimulus are impaired. Facts illustrating both forms of infrequent action, to all degrees between the normal rate and one of twenty per minute, are clinically known.

Whatever be its particular mechanism, an infrequent rate of beat may be attended with quick and abrupt or with slow and drawling ventricular contraction; and the radial pulse may, or may not, accurately reproduce the number and the qualities of the ventricular action.

286. (1) *Perverted innervation as cause.*—The tendency of concussion of the brain, of encephalitis in some cases, and of a variety of other cerebral affections, to slacken the rate of cardiac action, has long been practically recognised. Definite notions on the mode of connection between the cause and its effect have, however, been wanting: it has simply been presumed that by shock of, or altered circulation in, the nervous centre, the *exciting* force of innervation sent to the heart has been *diminished.* The site even of the deficiency of force has been matter of doubt,—the majority of observers localising this paralytic condition in the encephalon itself; while others think the deficient energy traceable to the vagi. Thus, Zengerke, finding that the pulse invariably slackened when the brain of the dog was pressed on with the finger (through openings made in the skull), refers the effect, not to the direct

pressure on the brain itself, but to that indirectly made to bear on the vagi nerves at the base. But he gives no conclusive reasons for the adoption of this view.*

The discovery of the *inhibiting* function of portions of the nervous system, as demonstrated by the experiments of Edward Weber, Pflüger, Lister, and others, naturally led to a revision of views on this subject. The result is that a converse doctrine has taken ground—namely, that *increased activity* of the *inhibiting* functions of certain portions of the encephalon (*corpora quadrigemina*), of the medulla-oblongata and of the vagi nerves is produced by the cerebral injuries and diseases referred to, and, through that increase, the heart's rate of movement slackened.

The mode of influence is supposed to be identical with that observed under galvanic excitement of the vagi. In speaking of the action of digitalis and of aconite on the heart we shall have occasion to consider this matter further.

(2) *Impaired cardiac contractility as cause.*—Inobedience to ordinary stimulus, and also incomplete or deficient readiness to contract, on the part of the heart, are shown in the infrequent action attending fatty metamorphosis of its fibre. But even in this case the nervous dynamism of the organ also is at fault; for no direct ratio holds between the amount of sarcous destruction and the slackness of the pulse. Mere weakness of the walls of the ventricles will not retard the frequency of their beat: in attenuated dilatation the pulse may be frequent; in the soft, flaccid, and weak states of the heart, occurring in typhus and typhoid, the action is invariably frequent.

287. Infrequent action of cerebro-spinal origin is strong; of cardiac origin weak: in either variety the heart may not beat more than from twenty to forty times in the minute.

288. There are other circumstances under which slackness of pulsation must probably be referred to the double cause. The peculiarly infrequent pulse of some persons, whose health is in all respects excellent, belongs to the number. So also does the slackened and enfeebled action that in certain exceptional cases marks the active period, or the convalescent stage, of acute diseases, attended with debility and nervous exhaustion; I have seen this well-defined in the acute stages of bronchitis, and during recovery from diphtheria, for instance: but, doubtless, the retarded beat is not specifically an attendant on the former of those diseases rather than on any other of a weakening character,—probably,

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* Ranking's "Retrospect," vol. xxvi.

however, it possesses specific significance in the latter, wherein I have known the pulse fall to 24 in the minute.

In cases where infrequent rate of beat is a physiological, and probably congenital peculiarity,* unattended with cardiac, respiratory, or cerebral disturbance, febrile diseases increase the frequency and strength of the action, and lessen any existent tendency to irregularity of rhythm.

(*b*)—OVER-FREQUENT ACTION.

289. The mechanism of increased frequency of the heart's pulsation, one of the most familiar phenomena of disease, is far from being fully understood. Beyond question the same rationale is not applicable in all cases.

290. Let the frequent pulse of pyrexia be taken by way of illustration. Upon which of the co-essential phenomena of febrile action does the accelerated beat depend? On the abnormal condition of the nervous system, which, if the teachings of Chomel be correct, forms the principal element of fever—involving a *plus* state of innervation of the cardiac plexus, or of the cardiac ganglia,—it cannot be a *minus* state of the inhibitory function of the vagi? On the rise in temperature of the solids and fluids,—a notion supported by the relatively exciting and depressing influences of atmospheric heat and cold? On the irritating influence of the blood (passing into the coronaries and playing on the endocardial surface),—altered as the fluid is in composition, both in the proportions of its normal elements, and by the accumulation of abnormal constituents, through defective excretion, excessive disintegration of tissue, absorption of morbific material from without, and septic changes within itself? To which of these influences alone, or in combination with others of the group, the accelerated action is immediately referrible, it is impossible positively to affirm.

Nor is our ignorance less of the precise mechanism, whereby spanæmia hurries the heart's action,—though changed blood, disordered innervation, and accelerated rate of breathing, all probably play their several parts.

Again, cases present themselves not unoften in practice, wherein without the slightest pyrexial rise in temperature, but in association either with general nervous disturbance or with local nervous disorder, for instance, of the laryngeal or gastric branches of the vagus, the heart's rate of motion increases greatly in frequency. In some such cases argument supports the notion of ganglionic stimulation, in others of diminished activity of the inhibitory power of the vagus, as the cause of the acceleration.

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* Vide an interesting case by Macdonnell, in "Brit. Amer. Journal of Medicine."

VI.—Action Perverted in Quickness.

291. Each independent beat of the ventricles may be effected with abruptness and quickness, or with deliberation and slowness. One or other of these conditions may exist, while the rate of successive beats is either frequent or infrequent. Both varieties are known clinically. The lagging, sluggish contraction of *some* hearts affected with hypertrophy is significant enough in diagnosis, when well defined.

292. Infrequent action, as already seen, attends a certain share of cases of fatty change of the heart: each action is then quick and short. Each contraction, long in coming, is brief in duration; the pulse is infrequent and quick.

293. In certain cerebral affections the ventricular action, lessened in frequency, is besides slackened in speed: the pulse is infrequent and slow.

294. Again, in many cases of non-rhythmical palpitation, the varying amount of quickness or slowness in the ventricular action adds an important element to the mass of irregularity.

295. The specific mechanism of this imperfection, where purely dynamic, is unknown; its relationship to fatty metamorphosis is glanced at with the history of that disease.

VII.—Cardiac Tremor.

296. True tremor appears from Volkmann's inquiries to depend on weakened tonicity of muscle; this tonicity in turn to be sustained by impulses from the spinal cord,—impulses successive, it is true, but so closely successive that the effect of any one has not had time to die away, before its successor has sprung into existence. Thus is explained a *constant* effect from an *interrupted* action. But the effect, like the action, becomes perceptibly non-continuous, if the impulses slacken in frequency. The result is tremor.

297. Now the occurrence of such tremor in the heart is even more problematical than that of any of the perversions of muscular action yet mentioned. Still the difficulty of accounting for the occasionally excessive irregularity both in *force* and rhythm of the

heart's action, justifies us in not at once rejecting any plausible hypothesis. It may be that certain forms of irregular fluttering palpitation in part consist of genuine tremor. That is, that (in addition to irregular movement arising out of mechanical difficulties and modified innervation of the heart's *contractility*), impairment and unsteadiness of the nervous force presiding over its *tonicity*, may by possibility contribute a share of feeble, wavering, non-rhythmical motion.

I have known this condition of fluttering palpitation suddenly follow an overstrain of the heart, and coexist with faintness and subjective sense of tremor all over the body.

298. Were this view sustained, the experiments of Volkmann (which prove that tremor of a voluntary muscle may be imitated by slackening the turns of the weak rotatory galvanic apparatus, so that the impulses may succeed each other at appreciable intervals of time) suggest by inversion a mode of treatment of fluttering palpitation.

§ II.—COMPOSITE DYNAMIC CHANGES, OR FUNCTIONAL DISEASES.

299. The clinical conditions, known as Functional Diseases of the Heart, are worked out of the elementary disturbances of cardiac innervation and motion, just reviewed, variously and capriciously associated. The list comprises simple palpitation or increased action; irregular action, without or with palpitation; decreased action and syncope; infrequent action; slow action; angina pectoris; and pseudo-angina pectoris.*

I.—Simple Palpitation.

300. By *simple palpitation* is meant increase in the force of the heart's contractions, unattended with any change of rhythm, and not necessarily with increased frequency of beat.

The amount of force may be slightly above the par of health; or sufficient to shake the chest or entire body of the patient, or even the couch on which he lies.

There is here the excess of force, induced as a *healthy* result of exercise, *morbidly* sustained by some centric, intrinsic, or reflex excitement.

301. *Symptoms.*—(*a*) Consciousness on the part of the patient of the heart's action seems, at the least, an almost invariable attendant on palpitation. A variety of attendant subjective sensations may be enumerated, occurring singly, in groups, or all of them combined. Such are choking sensations; a form of *globus*, or feel as if the heart were "jumping into the throat," and the eyes bursting from the sockets; præcordial anxiety; faintness, actual syncope, or partial unconsciousness. If præcordial pain exists, it commonly amounts to little more than a sense of dull, aching soreness; but in exceptional cases very severe pangs, similar to those of angina, attend the paroxysm: such pangs are sometimes relievable by pressure of the hand, even where the patient cannot endure the recumbent posture on the left side.

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* The student is particularly requested to refer back in studying each clinical functional disease to the description of the particular dynamic disturbances of which it is composed. His notion of those diseases would otherwise remain incomplete.

(*b*) Hurry of respiration, to any very notable amount, is of rare occurrence; it does sometimes, however, reach a degree out of proportion with the pulse, giving the patient the appearance of a person out of breath with running.

(*c*) Tinnitus aurium; vertigo and confused vision; cephalalgia, heat of head and flushed face; and clammy coldness of the extremities, add themselves to the list of disturbances.

(*d*) Although this be more common, when rhythm is perverted, yet even in attacks of simple palpitation there may be extreme general distress and fear of death,—singularly enough, more of this, often, than in cases of palpitation of organic origin: the consciousness of local suffering is rather in the ratio of the general and local sensibility, than of the actual palpitation.

(*e*) In some, but by no means in all, cases of long duration the eyeballs seem to enlarge, and distinctly protrude to an unnatural amount from the orbits, an enlargement giving more annoyance and apprehension to some sufferers than all the other evils of the complaint combined. This condition is, in truth, of great practical importance, and will be treated of apart as CARDIO-THYROID EXOPHTHALMOS [720].

(*f*) Vascular bronchocele, almost invariably coexistent with this protrusion of the eyeballs, will be considered in the same place.

302. *Physical signs.*—(*a*) The impulse is too extensively visible, but the apex-beat natural in site.

(*b*) If the heart be a well-nourished one, the impression it gives to the hand laid on the cardiac region, is that of a *blow*,—the impulse may even be somewhat heaving; if the organ be feeble, the impression is that of a *slap*. Systolic basic thrill may, under special circumstances, be felt [63, *a*].

(*c*) Habitually, the area of dulness remains unaltered, but extension to the right of the sternum may occur, in prolonged paroxysms especially; dulness is never carried upwards.

(*d*) The first sound is too loud and clear at the mitral apex, and somewhat abrupt and short; the second is lower-pitched and less clicking than natural at the mid-sternal base. The first sound may be loud enough to be audible, both to the patient and to by-standers, at a distance of some inches from the chest. Reduplication of the second sound at the base is common; and a distinct metallic ring, or a pericardial rub, may accompany the shock at the apex.

Will simple palpitation produce murmur in a structurally healthy heart? If there be the slightest coexistent spanæmia (so slight that in the state of ordinary action the sounds are perfectly normal), basic systolic murmur will most certainly be generated. But murmur of the same seat and time may also occur, both in males and females, during violent palpitation, where all attainable evidence points to the non-existence of any morbid state of the blood. I suspect that mere palpitation may also cause passing mitral regurgitant murmur of dynamic mechanism [p. 89], but of this I am not sure.

(*e*) The aorta, the carotids, and the arteries generally, beat with undue force, sometimes exceeding that of the heart itself,—more especially in spanæmic persons.

The radial pulse is quick, sudden, forcible (yet without real strength, for it yields easily to pressure), and over-frequent, when palpitation occurs in nervous, anæmic people. In the vigorous and plethoric, although the heart be perfectly free from over-nourishment, the beat may be full, hammering, heavy, and resisting: its frequency normal, or even below par.

The sphygmogram of the radial pulse in a well marked case of *simple sthenic* palpitation is distinguished by the verticalness of the line of ascent, and the height and uniformity of the line connecting the apices.

(*f*) Very slight jugular pulsation may, I think, occur independently of demonstrable tricuspid regurgitation, or of hypertrophous dilatation of the right ventricle.

303. *Duration.*—The heart of certain sufferers from palpitation is constantly in a state of more or less marked over-action: they are never perfectly free. But such cases are, on the whole, exceptional; the complaint is essentially paroxysmal. Lasting for a few minutes, an hour, or with remissions for days together, a fit of palpitation frequently terminates by sleep. In hysterical women a copious discharge of watery urine may occur at the time of release, but it is by no means a constant event.

The time, during which a patient remains subject to attacks, varies infinitely; as likewise the length of the intervening periods of freedom.

304. *Causes.*—(*a*) Early adult age, the female sex, and the nervous temperament are commonly held to *predispose* to palpitation. I do not deny the influence of any of these conditions; but the gravest examples of palpitation, disconnected with obvious organic disease, I have met with, certainly occurred in youthful males.

(*b*) The *exciting* causes range themselves under the heads of perverted innervation, morbid conditions of the blood, diseases of the lungs, and mechanical influences.

a. Perverted innervation may be centric, reflex, or intrinsic. The *centric* mechanism is exemplified by palpitation from emotion of various kinds, over-study, mental excitement, the abuse of alcoholic liquors and tobacco. To this category belongs palpitation attending certain centric nervous diseases, as hysteria, epilepsy, spinal irritation, chorea, hypochondriasis, and cerebral erethism with insomnia. So long as the rhythm continues regular and the action free from tumultuousness, interference with the inhibiting or co-ordinating function of the vagus cannot be held to exist. To *reflex* mechanism may be traced over-action excited by dyspepsia of all kinds; by certain articles of diet, as strong tea; liver disease; intestinal irritation, verminous or other; ovarian irritation and disordered menstruation; venereal excesses; masturbation and spermatorrhœa; and intercostal neuralgia. The play of the *intrinsic* nerve-centres, though difficult of proof, is probably not the less real. The observation already made [280, *foot note*] concerning the influence of those centres on rhythm will *mutatis mutandis* apply here.

β. Blood altered in composition, it is presumed, irritates the endocardial surface; if palpitation be so caused, the mechanism becomes really *reflex*. But it is also quite possible such blood, distributed through the coronary vessels, may directly irritate the nerve-centres,—the mechanism is then *intrinsic*. Be this as it will, spanæmia, plethora, uræmia, hyperinosis, hypinosis, gouty and rheumatic blood, seem all capable of inducing palpitation more or less actively.

γ. The influence of *pulmonary disease* is less frequently apparent than might be supposed. Still, the over-action, entailed by chronic bronchitis and emphysema on the right side of the organ, extends to the left, and constitutes a form of palpitation.

δ. To the *mechanical* order belong cases referrible to distortion of the chest, pressure from abdominal fluid or tumours, pregnancy, tight-lacing, sideward displacement of the heart by pleuritic effusion, &c.

ε. But inasmuch as every one of the conditions mentioned, external to the heart itself, *may exist without palpitation*, the probability is that in ultimate analysis the intrinsic motor nerve-centres or the sympathetic supplying cords are at fault, when those conditions are attended with palpitation.

305. *Treatment.*—The treatment embraces (*a*) the management

of the fit; and (*b*), during the intervals of seizures, the removal of the conditions exciting them.

(*a*) It may be possible sometimes to remove the cause of a fit at the time of its occurrence, as when palpitation depends on acid and flatulent dyspepsia. But in the majority of cases during its actual presence our exertions are directed rather towards lessening the intensity and shortening the duration, than removing the cause, of the seizure.

Thus the duration and amount of suffering may be diminished by free doses of antispasmodics, asafœtida, musk, and valerian, especially in hysterical females; by diffusible stimulants, ammonia, the æthers, and in some exceptional persons by very strong coffee; by narcotics and sedatives, opium, hyoscyamus, hydrocyanic acid. Acidity and flatulence, frequent causes of palpitation, may be corrected by soda and charcoal; a loaded stomach relieved by an emetic. If plethora of the sthenic kind be present, cautious venesection is advisable. Should a gouty or rheumatic state be discoverable, colchicum, guaiacum, and ammonia, and irritant stimulants to the joints, are the best remedies. Heat may be applied to the extremities, but the application of ice over the heart is a dangerous practice, especially if the rhythm of the organ be in the smallest degree affected.

Is *digitalis* an agent fitted to control a fit of *sthenic* palpitation? If it be held that the drug tells depressingly, *quasi*-paralytically on the heart's action, the answer must be unhesitatingly in the affirmative.* Careful clinical investigation, supported in some measure by experiments on frogs, as well as on warm-blooded animals, has, however, shown that the once-popular creed concerning the constantly depressing action of digitalis at least requires revision.

(*a*) *Experimentally.*—It has been found that in frogs systolic contraction, at first, sometimes increases in frequency† and perfectness under the influence of the drug,—while by and by each systole becomes prolonged, diastolic dilatation gradually growing less complete, as *portions* of the ventricle become firmly and persistently contracted from the apex upwards, until eventually general and complete stoppage of all action takes place in a state of firm ventricular contraction. In birds and in certain mammalia, the same result, as regards *post-mortem* appearances, has been observed.[*]

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* Even so late as 1861, Pfaff, after long and careful experimental and clinical inquiry, stated, among other of his conclusions: "The dose of digitalis must be reduced as soon as the *paralysing* effect is produced on the heart and arteries." "The *sedative* action of digitalis in marked excitement of the heart, is often perceived for seven or eight weeks." (Union Médicale, Fevrier, 1861.) [637.]

† F. B. Nunneley (Proceedings Roy. Soc., p. 48, June, 1869) found no increase in frequency or power in frogs poisoned by the hypodermic injection of one sixtieth of a grain of digitaline.

Death by digitalis would then be produced, as an inference from these experiments, by *tonic spasm*, instead of *paralytic distension* of the ventricle.

What is the cause of the observed increase in force of contraction? Does digitalis paralyse the inhibiting activity of the vagus [286], or intensify the motor energy of the sympathetic or the cardiac nerve-centres?

Traube espouses the former of these hypotheses. Having first ascertained that the pulse of a good-sized dog fell in sixty seconds from 132 to 24 beats per minute under the influence of eight grains of extract of digitalis, injected in solution into the jugular vein towards the heart, and that the frequency rose again in the course of about twenty minutes under the influence of four other injections, alike in amount, to 174 beats per minute, Traube proceeded to test the agency of the vagi in the matter by two separate series of experiments, in one of which the vagi were divided before, in the other after, the administration of the digitalis. The result was, the poison produced its effects more slowly, when the vagi had been divided. But, surely, section of the vagi must be at least equal in efficacy, as an inhibiting agent, to their paralysis by poison.

The second hypothesis, that of stimulation of the cardiac ganglia, or the intrinsic nerve-centres, is most generally accepted. It has been found that, if the heart of a cold-blooded animal be taken from the body, the rhythmical contractions which naturally continue for a time after the removal [284], are impaired in activity by a weak, arrested by a strong, solution of the drug applied to the apex of the organ.[†]

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[*] Traube, Dybkowsky, Pellikan, Hilton Fagge, Stevenson, Handfield Jones, Fuller, and Fothergill, take rank among the chief experimentalists; an epitomised version of their results may be seen in the essay of J. M. Fothergill (on Digitalis, 1871). But how far are these results applicable to man? Frogs, operated on by digitalis to the point of producing persistent spasm of their ventricle, do not seem for a while to be the worse for its fixed paralysis. Indeed, one experimentalist, most ardent in his advocacy of the applicability of these vivisection-results to the human subject, tells us, "the removal of the contracted heart by scissors did not cause the frogs any apparent inconvenience!" Where does this operator draw the line of separation between vivisection-results applicable and not applicable to man? Is he, perchance, a believer in the legend of St. Denis?

[†] Eulenberg and Ehrrnhaus, quoted by Fothergill, loc. cit. p. 16.

Neither theory can be said to be established; meanwhile the fact seems to remain that digitalis under certain conditions promotes complete ventricular contraction.

(β) *Clinically:* Digitalis (1) slackens the frequency of contraction; (2) relieves distressing sensations and so-called cardiac anguish; (3) gives firmness to systolic contraction, where, *under certain circumstances*, this has been distinctly wanting; (4) tends to regularise, and gives steadiness, with strength, to cardiac action, where the opposite conditions have obtained,[*] but it *does not do this in all persons under apparently identical circumstances* (*statical and dynamic*),[†] *nor even always in the same person in successive seizures of ostensibly the same nature;* (5) has not been demonstrated to do harm where ventricular over-action exists, provided cardiac distress be superadded; (6) if too long continued, and this even in cases where, at first, its use had proved sedative and invigorating, ends by *accelerating, disturbing the rhythm of, and depressing*, cardiac action; (7) in poisonous amount acts in two ways: if given in small doses too long, kills by carrying the last described effects to extremes; if given at once in excessive quantity (as in experiments on animals) kills apparently by spasmodically disturbing and arresting piecemeal the contractions of the heart.

To return from this digression to our starting point, is digitalis a fitting remedy in a fit of pure sthenic palpitation? Physiological experiment would appear to say, No! Clinical experience has always seemed to say, Yes. For my own part, believing that, in the case of a forcibly palpitating human heart, there must be conditions at work that cannot be represented in any form of experiment on a cold-blooded animal, I shall look to those conditions as explaining the apparent divergence of clinical results and of those of vivisection, and continue to employ digitalis. In its claims as a practical controller of excited action, one thing wholly forgotten by experimentalists, is the relief afforded to cardiac distress by the drug, the "opium of the heart," a relief which in turn promotes

[*] In a case of this kind, where, years ago (1867), I ordered digitalis in conjunction with R. Dunn, of Norfolk-street, great relief was secured—a relief suddenly exchanged for a state of the deepest cardiac suffering, in consequence of the patient having received a letter from a medical friend, warning him that if he persisted in taking digitalis he would assuredly die. He, however, took the digitalis in spite of adverse prophecy, practically regained health for a time, and is still alive.

[†] So certain is this, that the notion of a connection between special idiosyncrasy and the mode of action of digitalis has more than once forced itself upon me. Just as the influence of green tea on cardiac motion varies in different people, causing pure palpitation in the one, rhythmic disturbance in another, quietude of contraction in a third, while in a fourth its results on the heart, if any, are inappreciable, so the effects of digitalis inexplicably vary in various individuals.

a revival of healthy action, in whatever manner this may have been disturbed [637].

The dose of the tincture will vary from five to twenty minims, repeated with an amount of frequency proportional to the violence of the seizure.

Before dismissing the subject of digitalis it may be as well to observe, that there is nothing new (there *is* but little new under the sun!) in the notion, that digitalis acts most curatively on the whole, when the pulse is "feeble and intermitting," than when it is "tight and cordy,"—hence tonically, rather than depressingly: I include within inverted commas the words of Withering, written well nigh a century ago.* The sharpness of observation of men of that day, when the facts concerned lay within their range and means of appreciation, was just as great as of workers of the present hour: the abstract faculty of observing has not improved a whit. How often, too, are novelties nothing more than ancient opinions translated into modern phraseology. Withering seems to have thoroughly recognised that uncertainty in the action of digitalis, which it has just been suggested may, in some measure at least, depend on idiosyncrasy.

Aconite in *small* doses (not more than one to one and a-half drops of Fleming's tincture at a time) may be had recourse to, also, in the trust of lessening force and frequency of action.

Aconite and digitalis apparently act in precisely opposite manners on the frog's heart,—death is produced by the former through paralysis of the ventricle, and occurs in extreme diastole. Experiment would, indeed, seem to show that digitalis may act as an antidote to aconite, eventually contracting a ventricle which had undergone extreme distension by the latter drug: the converse power does not appear to be possessed by aconite in respect of digitalis.

W. Dobie reports a case (Brit. Med. Journal, Dec., 1872) where a man, having swallowed an ounce of Fleming's tincture of aconite, of extra strength, recovered after having had twenty minims of tincture of digitalis injected hypodermically, after galvanism had been applied to the cardiac region for twenty minutes, and after the administration of ammonia, brandy, and three teaspoonsful of digitalis within an hour. However interpreted, the facts are very remarkable. But the failure of the confident promises not long since made, concerning the antagonistic effects of belladonna in poisoning with opium, makes it wise not to accept too hastily the seemingly fair inference, that in this case the digitalis acted as the true antidote to the aconite.

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* Foxglove, and its Uses, p. 189. 1785.

There are numerous other agents which affect the heart's motion by contracting to excess, or annulling the contraction, of the ventricles, directly or indirectly, and which might be used in palpitation: colchicum, Calabar bean, veratrum vivide, caffeine, hydrocyanic acid, prunus virginianus, among the number. Colchicum will sometimes produce alarming depression, if given in very large doses, and I have had reason to relinquish the use of veratrum vivide from the same cause. Lead is too slow in action, marked though its influence eventually prove.[*]

Chloral is too uncertain in its influence on different patients to be trusted to. Possibly, as has been suggested, the rate at which chloroform is given to the blood by its decomposition varies in different people, a certain given rate being compatible with safety, a more rapid evolution of the vapour dangerous.[†] I have certainly known grave depression follow moderate use of the agent—say a dose of twenty grains, repeated twice in the course of twelve hours,—a quantity which would be taken by the majority of persons without striking effect of any kind.[‡]

(*b*) The measures to be adopted in the intervals of seizures will often vary in their essential feature with the nature of the constitutional or local fault that excites the paroxysms. But there are besides some general rules, applicable under all circumstances, which may be set down here. Persons subject to palpitation should avoid stimulants in any quantity, over-exercise, over-sleep, emotional and intellectual excitement. Hydrocyanic acid, aconite, digitalis, and belladonna, internally, varied according to circumstances (the latter also in the form of plaster over the heart), coupled with the use of the shower-bath, strict attention to diet, regularity of bowels, cheerful occupation, and, lastly, change of air, will either remove the tendency to palpitation altogether, or greatly mitigate the severity of the seizures. Should there be the least evidence of spanæmia, iron is indispensable.

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[*] In 678 of 1217 cases of lead-colic analysed by M. Tanquerel des Planches, the pulse ranged between 30 and 60.

[†] That chloral is decomposed in the blood into chloroform and formic acid appears now to be the accepted doctrine.

[‡] The quantity of chloral that may be taken at one time without grave results is yet undetermined. Habit will enable some persons and in some states of disease, to take enormous quantities with impunity. I saw a man suffering from chronic alcoholism (with C. C. Claremont), who had repeatedly taken *three drachms* for a dose, and ultimately, without any sensible hypnotic effect. He had a prescription for *one drachm* as a dose, and managed, by sending to three different chemists, to obtain what he considered his proper allowance.

A course of the bromides sometimes sensibly tranquillizes the organ.

II.—PALPITATION OF IRREGULAR RHYTHM AND FORCE.

306. This variety of palpitation is in its extreme forms essentially paroxysmal: persistency of its highest manifestations and continuation of life would be, in point of fact, incompatible.*

307. *Symptoms.*—A typical paroxysm is characterised as follows. *Motorially:* the action of the ventricles is unsteady, hesitating, or anticipating; intermittent at regular or irregular intervals; the force of contraction now increased, now diminished; the sequence of the auricular and ventricular contractions irregular; the synchronism of both ventricular, as also of both auricular, contractions lost; the valvular and papillary actions more or less perverted in rhythm; the rate of motion perpetually changing; and associated with all this, probably, in some cases, genuine tremor of the organ [296,—*vide* also 103, *i*]. *Sensorially:* combinations in all conceivable varieties of cardiac paræsthesiæ and hyperæsthesiæ,—making up in their totality various amounts of so-called "præcordial anxiety."

A sort of order in disorder is sometimes observed,—a regular recurrence of certain conditions of irregularity taking place at fixed intervals; in these rare cases cycles of irregular action exist.

308. The radial pulse of course shares in the rhythmical disturbance. The precise relationship is, however, very difficult to trace; the weaker class of systoles constantly fail to impress the artery at the wrist. This matter has, however, been already considered [103, *i*]. The rate of pulsation at the wrist may, in one and the same case, be *momentarily* marked at fifty in the minute, the next instant rising to a hundred and fifty, or becoming uncountable from combined frequency, irregularity of rhythm, and inequality of force.

The frequency of breaks in regularity varies greatly. A patient who for a considerable time kept an elaborate register (of which a sheet is now before us) of the number occurring in four consecutive minutes at different times of the day, found them range between one and forty-five: the latter number after a "light dinner with champagne;" the former after "soup, fish, andsherry."

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* Pure intermission of the pulse at regular intervals, fully considered elsewhere [103, *i*], is not referred to in the present section: it can scarcely be considered, clinically speaking, in the light of a disease, when demonstrably disconnected with textural change, cardiac or other.

The sphygmogam of the radial pulse translates all this in variation of verticalness of the line of ascent in different waves, in polycrotism of the line of descent varied with dicrotism, in great irregularity of the lengths of the line of ascent and of descent of each wave, and total irregularity of the lines joining the apices and the bases of a series of curves [211].

309. Syncopal tendency, or actual passing syncope; pallor of face, alternating it may be with lividity; breathlessness; thirst; nausea or vomiting; disposition to relaxation of the bowels; haggard and anxious countenance; intense general uneasiness; jactitation; alarm, even fear of death; and clammy perspiration, mark at once the reflex suffering of allied organs, and the deep sympathy of the frame at large with the disordered function of the heart.

310. Immediately on the close of an attack, copious watery flux from the kidneys sometimes occurs,—and this not merely in frail, nervous, hysterical women, but in strong men. In the latter especially micturition may be distressingly frequent.

311. *Physical signs.*—(*a*) The impulse may be slight in amount, fluttering in character; or irregularly forcible, now strong, now lost, both to the eye and hand; in extreme cases the motion becomes jogging, trembling, rolling, quivering, or indescribable.

(*b*) A heart beating, now fifty, now one hundred and eighty or more times in a minute, now with excessive force, the next moment with such feebleness that the hand scarcely catches the impulse, cannot fail to become more or less clogged and obstructed internally. The evidence of this appears not only in the syncopal and suffocative tendencies of such fits, when at all prolonged, but in the increased area of the heart's dulness,—especially to the right of the sternum.

(*c*) The rhythm of the sounds is, of course, altered,—reduplication is common [103, *k*, 2]. Their strength and quality vary from moment to moment,—the general tendency of the first of the two, especially, is to shortness and undue sharpness of tone: but occasional forcible contractions may give a healthy, or even an excessive, systolic sound, masked possibly by parietal shock or pericardial rub.

On the occasional production of dynamic murmur by palpitation I have already dwelt sufficiently. But it may be well to observe here that, conversely, pre-existent murmur may be covered either partially or wholly by violent shock-sound during a paroxysm of excessive action.

312. *Causes.*—The extrinsic causes of palpitation of this form are the same as those of the simple variety. The intimate mechanism of irregularity has already been considered [280]; the intrinsic conditions in the heart and its appendages which bring that mechanism into play—which cause a given heart to palpitate irregularly rather than regularly—are not only beyond our ken, but defy our present means of investigation. Palpitation of irregular rhythm is more prone than the simple variety to occur from blood-alteration [304, β]: this is particularly true of gouty blood-changes. So, too, it has occurred to me to meet with palpitation and distress of this form, and this form only, as a result of exposure to *paludal* poison.

313. *Prognosis.*—The prognosis is, absolutely speaking, serious; and relatively much more so than in the previous variety. Mere irregularity of rhythm, if extreme, cannot be regarded as a matter of light importance,—notwithstanding exceptional individuals exist, who, by some peculiar idiosyncrasy, enjoy good health, though their pulse be more or less irregular [282]. But in these persons, the organ, though irregular in its beat, is normal in force of action; clearly the case becomes a very different one where palpitating motion is added to irregularity. And though by possibility the combination may exist without structural change, I confess I have rarely encountered it, where there was not either certainty or strong suspicion of alteration in the heart's texture. At all events, I am persuaded that when elderly persons are the frequent subjects of what is called "stomach-palpitation," the assumption that, because no valvular signs or evidences of marked hypertrophy or dilatation are discoverable, therefore the heart is sound, is a positive error. There may exist (as demonstrable post-mortem) soft flabbiness of texture or incipient fatty metamorphoses, which clinically gives no appreciable sign. Death may, under such circumstances, actually occur during the fit.

314. *Treatment.*—In treating a paroxysm of this, as of the previous, form of palpitation, the search after causes must never be omitted: and that search is even more important as a guide to management in the intervals.

(*a*) Stimulants and antispasmodics are the agents most efficacious in this form. Among the former ammonia occasionally acts with great promptitude; but the *facile princeps* is brandy, from the facility with which it may be obtained. The quantity to be given as a dose will vary with the intensity of the paroxysm, the amount of depression and the habits of the patient *quoad* alcoholic drinks. From a teaspoonful to half or even an entire wine-glassful, are the ordinary extremes. Under their influence the beat will sometimes acquire comparative steadiness in the course of a few minutes, and the rate of motion notably slacken.

If there be local pain, opium should be freely associated with the stimulant administered.

Digitalis may be employed, especially if the rate of movement be very rapid, with relief to the cardiac suffering and a slackened, steadier action. We may expect this issue, even where the attack is of the gravest aspect, and where the irregular and violent action is connected with extreme aortic reflux. The agent may be given in doses of twenty minims of the tincture, and suspended before producing any very marked diminution of the heart's beats,—both from the fear of causing stasis and thrombosis in the right cavities, on the one hand, and from the possibility of violent reaction.

Mustard poultices to the præcordial surface seem, in some cases, to help in regulating the heart's action; they lessen local distress, and never do mischief.

(*b*) In the intervals, iron, and other metallic tonics, the use of the shower bath, and an invigorating regimen generally are indicated. Where this can be effected, change to a climate at once mild and bracing should be tried. Walking exercise under favourable climatic conditions sometimes proves very serviceable, where no structural change has occurred in the heart: a pedestrian excursion in a mountain district has, under such circumstances, proved, within my knowledge, singularly restorative.

Soberness in the use of alcoholic stimulants is even more important in this form than in the simple sthenic variety. The free use of wine and spirits in the intervals of seizures not only renders the patient more subject to attacks, but deprives him of one of his chief aids during their occurrence. I am the more earnest in insisting upon this, from having known more than one case of chronic alcoholism (in females especially) originate in the use of brandy for the "prevention of palpitation." On the other hand, moderate indulgence in wine need not be discontinued,—experience alone will teach the sufferer what kinds may be taken with least discomfort. Thus some men, who cannot swallow a single glass of the driest champagne without palpitation ensuing, readily take a pint of sparkling Schartzberger (Moselwein) without the slightest inconvenience of the kind. I think there can be no doubt about the propriety of ostracising malt liquors from the table of any one subject to palpitation of irregular rhythm.

Yet more certain am I that tobacco, whether chewed, snuffed, or smoked, exercises a most depressing influence on such hearts, and tends to disturb their rhythm. Whether the older physiologists were right in ascribing these ill effects to nicotine, or whether they be really induced (as affirmed by Vohl and Eulenberg *) by pyridine, picoline, colledine, and other bases produced during the combustion of the tobacco, the practical result is the same—tobacco is to be avoided.

III.—Palpitation of Regular Rhythm and Force, but of Increased Frequency.

315. There is a form of palpitation marked simply by excessive frequency, without increase in force or alteration of rhythm, of the heart's beat. Here we have a simple dynamic perversion carried to such a height as practically to constitute a disease [289].

316. *Symptoms.*—In addition to the increased frequency of action (the pulse may reach 100, 120, 140, 160,—while I write this page, an example of the latter rate has passed under my notice) there may be various cardiac paræsthesiæ [263], and occasionally downright pain. Occasional faintness is a tolerably common accompaniment. I am inclined to believe dry pericarditis may be brought on in peculiar states of the system by sustained over-action of this kind.

317. This is the variety of palpitation most common in sufferers from enlargement of the thyroid gland with exopthalmos [720], though often holding on for weeks and months independently of any such association.

318. *Causes.*—As far as I can remember, this form of palpitation has never fallen under my notice in a male. By no means peculiar to hysterical or "nervous" people, it may occur in women distinguished by force of character, and so-called strong-mindedness. In three instances theomaniac tendencies were exhibited.

The exciting causes have been pedestrian excursions, ascent of mountains, acute pain with effort to control its manifestation, prolonged mental distress; in not a few cases of the class the cause baffled inquiry.

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* Brit. Med. Journal, May, 1872.

319. *Treatment.*—Digitalis has little influence on this form of palpitation—at least (I can answer for this), given in ordinary small doses. Nervine tonics and general measures for improving the health alone seem really serviceable.

IV.—Decreased Action and Syncope.

320. Decreased action of the heart, affecting force and frequency of contraction, if carried to extremes, produces syncope. In its minor degrees, and if an habitual state, it entails general languor of the functions. Let us first consider the phenomena in their extreme degree.

A.—SYNCOPE.

321. Syncope, in its most perfect condition, is a state of apparent lifelessness, in which failure of the heart's propulsive action forms the first link in the chain of events tending to somatic death. The second link, suspension of the functions of the encephalon, is followed, almost accompanied, by the third, cessation of breathing.*

322. *Causes.*—(*a*) The conditions which appear to *predispose* to syncope are the female sex, early adult age, nervous and especially hysterical temperament, spanæmia, general weakness and idiosyncrasy. Cases every now and then occur in which the importance of the latter condition appears paramount. I have met with a fair number of persons in the course of my experience who, perfectly sound in all their organs, were in the habit of fainting, sometimes without the intervention of any traceable exciting cause, sometimes under an influence so slight that in the mass, even of feeble and nervous women, it would have proved wholly incapable of arresting the actions of the heart. Doubtless in such persons there must be some inherent weakness or specific impressionability either in the cardiac plexus, ganglia, or nerve-centres, or in the connected centric portion of the nervous system,—or in the irritability of the muscular structure.

(*b*) The *exciting* causes of syncope, excessively numerous and diversified, may be arranged under three heads, *dynamic*, *hæmic*, and *organic*, including *mechanical*.

a. *Dynamic and proximate.*—The cause of stoppage here is either failure of the special irritability of the heart, paralysis of its motor nervous apparatus, or spasm of its fibre.

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* The student is requested to read as an essential addendum to this account of syncope, the section on Motor Paralysis of the Heart [268—272].

(1.) The experiments of Blake would tend to show that the salts of potash, magnesia, zinc, copper and lime, injected into the blood, destroy life by specially annulling cardiac muscular irritability. Brodie, many years ago, drew the same conclusion as to the mode of action of the essential oil of tobacco in inducing syncope, while the nicotine he supposed to act on the cardiac nerves. Possibly here, too, might be ranked some examples of fatal fainting, where the heart is fattily metamorphosed, and where no rupture or intracardiac thrombosis has occurred to complicate the problem.

(2.) Paralysis of the nervous apparatus may be *centric*, as from direct injury to the brain in severe concussion,—an effect harmonising with the results of Legallois' experiments, which show that, though the heart continues to beat after gradual and piecemeal removal of the entire brain, its movements are arrested by sudden crushing of the encephalon.

Intense emotion of all kinds, pain, the sight of injuries inflicted either on oneself or on others, occasionally arrest the heart's beat.

Or the arresting influence may play on the *vagus nerves directly supplying the heart*, as in fatal syncope from excessive galvanic stimulus [328, *c*], and from lightning. Slight syncopal symptoms may be induced by irritation of the nerve,—and the fact has even been utilised in surgical practice to aid in the reduction of dislocations.[*] In all these cases the excitement of the nerve intensifies its inhibiting function.

Or the influence may be *reflex* through the sympathetic, as shown in fainting, caused by a blow in the epigastrum violently impressing the semi-lunar ganglia and solar plexus. Or reflex through specific impressions on the nerves of sense, as in cases of fainting from the influence of particular odours, or, in individuals of exquisite musical organisation, from the nerve-jar produced by continued false notes impressing the ear.

Or the impression may be reflex through the spinal nerves. Instances in point are: fainting caused by the movements of the fœtus in utero, acting through the uterine nerves; indigestible food,[†] or a draught of cold water taken during perspiration, or excessive hunger and nausea, acting through the gastric nerves;

[*] A. Waller, Proceedings of the Royal Society, May, 1870, p. 342. Here the method employed was compression on both sides of the neck, and no effect was observed, till the pulsation of the carotids had ceased to be felt.

[†] In part, however, this may be from the mechanical effects of distension with gas. New bread rapidly eaten, especially if the stomach has been for some time empty, may actually induce fatal syncope. Every one knows the painful story of Otway's death from ravenously eating a roll, when in a state of starvation.

injuries to the surface, as burns, through the cutaneous nerves; the introduction of a bougie, through the nerves of the urethra,—all of them occasional causes of syncope.

Or the paralysis may come of failure of the ganglionic motor nerves,—this is probably the origin of the syncopal distension of the ventricles in poisoning by aconite.

(3.) Spasm of the ventricle acts, under some circumstances, as the cause of syncope, when induced by digitalis.

The cause of syncope in Angina Pectoris will be inquired into with the history of that affection. The mechanism of fainting from prolonged stay in the warm bath is probably complex. The prolonged syncope which sometimes puts a close to the lingering career of phthisical patients, it is suggested by Louis, may depend on "fatigue of the heart,"—a phrase apparently equivalent to failure of irritability.

β. *Hæmic.*—In this category appears loss of blood, acting at once *directly* on the heart in consequence of deficient immediate stimulus to the ganglia, nerve-centres, and tissue of the organ, and *indirectly* in consequence of the impaired state of general innervation. Here rank, too, the circulation of imperfectly oxygenised blood, as where syncope occurs from non-ventilation in crowded rooms; and draining away of the water, as of other elements, of the blood through profuse discharges, diarrhœal or other. Under this head may also be placed cases of fatal syncope from thrombosis within the pulmonary artery,—forming a link with the following group.

γ. *Organic and mechanical.*—Here may be ranked cases of fainting from pressure of blood thrown into the pericardium from rupture of the heart, or of an aneurismal sac (the nervous shock of the injury playing a part, too, in the result); from disturbed circulation depending on ruptured valve, or even on old-standing organic disease of a valve,* or on sudden obstruction of a coronary vessel; from the entry of air into the circulation; from the sudden removal of extra-pressure, to which it had long been habituated, from a portion of the vascular system, as when fainting follows the incautiously rapid removal of ascitic fluid.

δ. As matter of actual experience, syncope is oftener induced by a combination of different causes than by any single one of the number. Fatal failure of cardiac action during the administration of chloroform, in cases where somatic death is very immediately effected through the heart, may be cited as an apposite example. In the first place it cannot be questioned, it appears to me, that in the mass of cases of actual death, or of imminent unachieved death, from chloroform, saturation of the nervous centres, for the tissue of which the hydrocarbons seem to have a special affinity, is the essential and radical cause of the event. Now the medulla oblongata being poisoned, its inhibiting function and that of the vagus become excited, while the heart's texture being supplied with blood largely impregnated with the inhaled vapour, it follows that anæsthesia of the cardiac ganglia and of the cardiac plexus, with impairment of sarcous irritability, are readily conceivable, nay, necessarily produced effects. Yet to all these combined conditions death cannot in ordinary cases be referred,—for the respiration in healthy animals invariably, or all but invariably, stops before the pulse,—the death is asphyxial not syncopal. It is easier to kill through the medulla oblongata by arresting respiration, than by directly or indirectly annihilating the heart's motor innervation and irritability. Some further influence is wanted, in order that the common sequence of failures shall be inverted, and that cessation of all action in the heart shall precede that in the lungs. Now that further influence is to be found, in the human subject, either in pure and inexplicable idiosyncrasy; or in pre-existing perversion of cardiac innervation; or in actual disease, most commonly fatty metamorphosis of the sarcous or nervous structures of the heart. These seem to be the conditions which occasionally convert paralytic suspension of cardiac movement into the second instead of the third element of somatic death. Nay, more, it is well conceivable that in some instances, where the heart is in a dynamic point of view very exceptionably impressionable, or where its own proper nervous structure has been extensively disorganised, its movements shall be arrested, before even the nervous centres themselves have given evidence of functional disturbance. Death would then be primarily and truly syncopal.

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* The only example, strictly in point, of this kind is furnished by the fatal syncope of aortic regurgitation,—the mechanism of which will be more fully considered hereafter.

323. *Anatomical Characters.*—Does death by syncope entail, as matter of necessity, any particular state of organs, distinctive of itself, and constituting its anatomical character? I believe this question must be answered in the negative. The mechanism of syncope is so various, its accomplishment sometimes so thoroughly instantaneous, sometimes, relatively speaking, so gradual, that the walls and cavities of the heart cannot be expected to, as they actually do not, present invariably the same

characters. In one case syncope is preluded by feebleness, in another by spasmodic force, of contraction: one individual is gorged with blood, another is anæmic, at the moment of the heart's last beat.

Hence it is, that in some cases the muscular structure is thoroughly flaccid, the cavities large and gaping; in others the walls firm and rigidly contracted, the cavities small and closed,—the heart's last effort terminating in ventricular systole. So, again, the cavities may be empty; they may contain a small quantity of blood, fluid, grumous or fibrinous; they may contain good quantities of grumous blood. Such blood as is present, is pretty equally distributed to both sides of the organ, or there may be an excess on the right,—though never to the degree seen in death by genuine asphyxia. There may or may not be fluid blood or actual fibrinous clot in the pulmonary artery,—of course, it is meant, exclusive of cases where actual thrombosis in the vessel is the cause of death.

Bichat taught that when syncopal death occurs in the course of various diseases indiscriminately, the lungs are "as a constant rule almost entirely free from blood."* He ascribes this freedom of the lungs to sudden cessation of the circulation; but in point of fact the cessation is not always absolutely sudden. There is time allowed for stagnating blood to accumulate in the pulmonary vessels. And so, as matter of experience, some slight engorgement may be found occasionally in perfectly healthy lungs after syncopal death; never, however, that massive engorgement of capillaries and fine vessels appertaining to asphyxia. Thus, in the remarkable case of a gentleman, who died by instantaneous syncope while in the act of signing his name (the signature was about half completed) to a few testamentary words, there was con-considerable engorgement of both lungs. Here, however, chill had had distinct influence on the death, though the patient died in his bed.†

The encephalon, unless under circumstances wholly exceptional, is pale and bloodless.

324. *Symptoms.*—Syncope, whether induced directly by failure of the heart's irritability, or indirectly through deficient supply or perversion of nervous influence, by loss of blood, or by me-

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* Vie et Mort, p. 189, 3ième édit. 1805.

† This will became the subject of proceedings in the Court of Probate; where it was held, the deficiency of witnesses' signatures, and *not* the incompleteness of the testator's own signature, deprived the document of all legal significance.

chanical causes, occurs with very much the same train of symptoms when actually and completely developed. But in respect of premonitory occurrences and the sequence of symptoms, differences occur according to the nature of the cause.

(*a*) *Premonitory.*—The more important of these are: nausea, sinking feel at the epigastrium, disturbed vision, vertigo, tinnitus aurium, confusion of thought, pallor, drawing of the features, inclination to clammy perspiration, tremulous contraction of the muscles, or slight convulsions, chattering of the teeth, and failure of the pulse. One or more of these may announce the coming syncope; but in a certain proportion of cases the actual stoppage of the heart's action is sudden, or perfectly instantaneous and unpreluded.

(*b*) *Actual.*—As the syncopal state grows more complete, the pulse, though absolutely wanting at the wrist, may often be faintly felt in the carotids; the patient becomes totally unconscious; the surface cool, clammy, or natural in feel and temperature; the features contracted; the nares pinched; the lips of marble pallor; the face and skin generally more or less blanched; the respiration suspended completely, or almost imperceptible; the pupils dilated: in some few instances the sphincters of the bladder and rectum relax.

Meanwhile the heart's impulse may be almost or completely lost to the eye and hand; the sounds nevertheless may still remain distinctly audible; both are, of course, exceedingly feeble, the first very short, the second generally lost at the apex. When the heart's motion wholly ceases, and the syncopal state is carried to its extreme point, no vestige of sound can be caught. Absolute suspension of cerebral* and of pulmonary action follows.

Pre-existing murmur may cease to be audible, though the heart's sounds continue perceptible [222].

325. *Duration.*—Lasting for an instant only, for seconds, or for a *very few* minutes, ordinary syncope terminates by gasping or rather sighing respirations, at long intervals, and gradual return of pulse, consciousness, and colour. Sometimes vomiting or discharge of flatus, convulsions, palpitation, or profuse perspiration, take place at the time of returning consciousness. Various paræsthesiæ may be felt in the limbs, and the phenomena of re-establishment of somatic life are on the whole generally represented by persons, who have actually felt them, as painful and dis-

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* The ancient synonyms of syncope, *deliquium animi* and *leipothymia*, show the importance of this element of the symptomatology.

tressing. Yet not always so, if we may trust the quaint and picturesque description left us by Montaigne of his recovery from fainting caused by a fall from horseback. . . . "Mais à la vérité non seulement exempte de déplaisir, ains meslée à cette doulceur que sentent ceulx qui se laissent glisser au sommeil. . . . Je prenais plasir à m'alanguir, et à me laisser aller." (Essais, liv. ii. chap. vi.) In the same chapter the acute essayist expresses the opinion that in all probability the moment of passage from life to death is unattended with travail or suffering of any kind,—an opinion much better in accordance with serious observation than that upheld by professional theosophists, who are eloquent concerning the sharp pangs that precede death, and the agony attending the separation of soul and body. Infinitely more likely to be true, judging even from my own clinical observation of multiform modes of dissolution, amid surroundings the most varied (in poverty and in wealth; in youth and in age; in men and in women; in the peaceful lull of emotional quietude, and in a state touching on the border regions of despair; in wearied indifference to all the evils and all the goods of life, and in a spirit of clinging devotion to some loved thing on earth; lonely, among strangers, and caressed to the last by relatives and friends; in faith the strongest and in utter disbelief of any known creed),—infinitely more likely to be true, that the passage from life to death is accompanied with pleasurable instead of painful sensations. And, in bearing this faithful witness, I merely lend support to the doctrine concerning the sensational accompaniments of the act of dying held by Cicero, Seneca, Lucan, Barthez, and many other medical and non-medical philosophers.[*]

It would be difficult, if not impossible, to fix with certainty the length of time the heart may fail to give signs of action either to the hand or ear of the observer, without necessarily ceasing to beat for ever. But I do not believe there is any record of trustworthy character proving that its action has ever been re-established after all cardiac sound had ceased for five minutes.[†]

326. *Diagnosis.*—(*a*) Syncopal unconsciousness will be distinguished from that of *asphyxia* by the pallor of surface; in the

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[*] On the whole subject the curious reader will find some very interesting matter collected in that strangest of books, "Le Lendemain de la Mort," by Figuier (4ième Edit., p. 400).

[†] Josat peremptorily affirms, that the absence of auscultatory signs of cardiac action, even if "prolonged for some minutes," is not an "infallible sign of death." ("De la Mort et de ses Caractères," p. 81.) But the real question is, what does the little word "some" mean?

latter state congestion and general lividity of the head and face exist, and the heart continues, though feebly, to beat.

(*b*) In *uræmic coma*, or semi-coma, there is pallor of face to deceive, but it is an œdematous pallor; the odour of the breath is urinous; and, finally, the state of the heart's sounds and the fulness of the pulse at the wrist will prevent error.

(*c*) *Apoplexy*, though traced *post mortem* to cerebral hæmorrhage, sometimes occurs with pallor of face; the state of the pulse and of the heart prevents the possibility of error,—in apoplexy, neither heart nor pulse fails in strength, and both may act with undue energy.

(*d*) *Hysterical Trance or Lethargy*, with absolute motionlessness, closure of the eyes, almost complete suppression of respiration, simulates syncope; but there is no pallor, and the pulse beats steadily and with distinctness.

(*e*) The only serious difficulty consists in diagnosing with certainty that rare form of prolonged syncopal trance, in which *somatic, and ultimately even molecular, death* may be closely simulated.

The alleged characters of real death may be enumerated as follows. Absolute immobility of the body; cooling of the surface; muscular (cadaveric) rigidity; opacity of the tips of the fingers, examined opposite a strong light; lividity and staining of dependent parts; an odour *sui generis;* absence of moistening of the mouth after it has been artificially dried; relaxation of the sphincters; the existence of higher thermometric temperature at a certain depth within the bowel than on the surface;* total absence of cardiac sounds; absence of breath-mark on a glass held close to the mouth; non-formation of blebs under the action of boiling water dropped on the skin; † non-appearance of blood from incision of a mucous membrane; insensibility of the muscles to the galvanic current; state of flexion of the thumb within the flexed fingers; flattening of the cornea; change of form of the pupil, from the round to the elongated, by lateral pressure on the eyeball (Ripault); dilatation of the pupil with insensibility to the galvanic current; disappearance (beginning with the deepest) of the three images, which are during life visible in the depths of the eye, if a lighted candle be held before that organ (Legrand); the non-oxidation (even at the end of half-an-hour or an hour) of a needle inserted into the flesh (Laborde).

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* For the account of an instrument invented for ascertaining this character by Van Hengel, vide "Gaz. Médicale de Paris," 1848.

† It appears certain, however, that blebs will sometimes form after real death. Martinot, of Lyons, affirms that a *dry* vesicle may form after death, under the flame of a candle, but never a *liquid* one.

Many of these characters will be useless *at first;* that is in determining whether on the *instant* of their occurring (and in certain legal aspects this may be all-important) the phenomena before us are those of syncope or of death. Besides the conscientious analysis of Josat (*op. cit.*) has shown that no single one of these characters is worthy of implicit trust; but in proportion to the number of them associated in any instance, may the certitude of decease be assumed. Still in doubtful cases, before a positive opinion is hazarded, sufficient time should be allowed for the development of putrefactive changes; lest a catastrophe, one of the most horrible the mind can conceive, occur.

The feats of the Hindoo Fakirs, who it is averred bury themselves alive, and exist in a state of "self-hypnotism" for a considerable number of weeks without oxygen, seem to me to rest on no trustworthy evidence. Even were disinterested witnesses prepared to vouch for the fact, I should avail myself of Hume's argument concerning miracles in general, and prefer believing that a certain number of honest persons had been deceived, than that the laws of nature had been reversed. The raising of a mango-tree from the seed in a single hour—stem, leaves, flowers, fruit—a prodigy daily accomplished by Hindoo operators, is a sufficient evidence of the remarkable powers of deception possessed by these people.*

327. *Prognosis.*—In the immense majority of cases syncope is recovered from, and without sequential ill-effects. Nevertheless, if chance brought the physician in presence of an attack of syncope in a person of whose medical history he knows nothing, and more especially if that person were no longer young, a cautious opinion as to the issue should be given. Organic disease of fatal tendency may in truth be present, but prove undiscoverable, on account of the heart's *quasi*-inaction, or but feeble effort at pulsation. It is affirmed that syncope has sometimes terminated fatally in cases of pure anæmia; and inasmuch as I have seen such anæmia end by death, I can well conceive that its immediate mechanism may be syncopal.

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* The exact manner in which the jugglers proceed is this. Having had the ground to themselves all the previous night, they make their exhibition the following day. First they sow a seed, plant it, put a great basket over the ground, remove the basket in so many minutes, and show the plant growing,—cover again, remove again, and show the plant with the leaves,—cover again, remove and show it with flowers,—finally, once more repeating the process, exhibit it with fruit.

On the other hand, syncope, instead of destroying, proves the very means of preserving, life under certain special circumstances. The fatal termination of grave hæmorrhages, traumatic and idiopathic, is very frequently prevented by failure of the heart's action giving the necessary time for the stagnating blood, within the bleeding vessels, to coagulate and close their opened orifices.

328. *Treatment.*—(*a*) In treating an attack of syncope, the first measures are to place the patient horizontally with the head on, or below, the level of the shoulders, to allow a free circulation of cool air, and remove all pressure from the neck and chest. If the syncope be caused by loss of blood, a tourniquet may be applied with advantage to one or both femoral arteries. Stimulant impressions on the nerves,—on those of the nostrils and lungs by ammonia, strong acetic acid, the fumes of burning feathers,—on those of the skin by the cold-water dash, the application of vinegar to the temples, slapping the palms of the hands or surface generally, frictions with stimulant liniments along the spine,—on those of the stomach (if the patient be sufficiently recovered to swallow), by a draught of cold water, frequently arouse the heart instantaneously. Æther, aromatic spirits of ammonia, or brandy, should be given internally,—if possible, by the mouth; if this be impossible, and the fit be prolonged, enemata, containing ammonia, turpentine, or brandy, may be administered. It is scarcely necessary to say, that if protracted syncope depend on an overloaded state of the stomach, an emetic should be given (by the rectum, if otherwise impossible) : flatulence may be relieved by the rectum-tube, and an asafœtida and cajuput enema.

(*b*) In cases of protracted fainting, assuming the serious character of suspended animation, hot applications, sinapisms or turpentine fomentations, to the heart and spine, electro-galvanism, and artificial respiration, must be successively had recourse to. If the cause be loss of blood, transfusion presents itself as a final measure. The last three agents require special consideration.

(*c*) Valentin, Henle, Budge, and others, have distinctly shown that the heart's action may be re-excited after stoppage by galvanic stimulation of the roots of the spinal accessory nerve, and of the first four cervical nerves, and of the sympathetic filaments belonging to the organ. Schiff states that increase of the heart's vigour takes place under very gentle stimulation of the pneumogastric. The careful experiments of Lister* seem to establish the general conclusion, that very mild galvanic currents passed from the posterior region of the spine, in the course of the vagi, and through the cardiac sympathetic excite, while strong currents exercise an "inhibiting" influence upon, the heart's actions. But when exhaustion has occurred in the animal experimented upon, a much stronger current is required to stimulate the heart, than when its nervous energy is scarcely or not at all impaired.[*]

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* Royal Soc., Aug. 13, 1858.

The practical guidance seems clear as to the force of galvanic currents to be employed in cases of protracted syncope in the human subject. The gentlest amount of galvanic stimulus should first be tried: but it is plain, if there has been much previous exhaustion, a powerful current, which under ordinary circumstances would have arrested the heart's play, may be required to revive it. Caution is obviously of deep importance; Brown-Séquard is doubtless correct in his strong assertion: "the best means we possess for *destroying* the remains of life in dying persons, is to subject their nerves and their muscles to the *exciting* and *exhausting* action of galvanism." So, too, a Committee of the Société d'Emulation de Paris found that electricity, employed as a general nervous stimulant, rapidly exhausted the nervous excitability of animals in the last stage of chloroformisation.[†]

(*d*) Transfusion of blood has been mentioned as a final remedy when suspended animation depends on excessive hæmorrhage. The propriety of the measure under these circumstances is self-evident. But, possibly, even in prolonged syncopal trance of other mechanisms, the injection of blood might stimulate the vaso-motor system of nerves, and so act on the heart. The marvellous effects of saline injections in cholera Asiatica, suggest the possibility of their proving yet more useful in syncopal loss of animation. Again, a patient, quasi-dead from cholera, may be temporarily resuscitated by the injection of a fluid imitative, in temperature and saline constitution, of the water and salts of the blood: true, in the great majority of cases he eventually perishes; but here there is the disease to destroy—in the case of revival from mere trance there is none.

(*e*) In employing artificial respiration gentleness is of the last importance. I long since pointed out the danger of producing interlobular emphysema by too forcible insufflation.* Force is fatal. And in truth the efficacy of artificial respiration in genuine syncopal trance must be limited enough. It is another matter, where syncope is secondary to asphyxia, as in cases of imminent death from chloroform-inhalation.

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[*] Waller, while insisting on the "collapse and syncope, and stoppage of the heart's action," produced by *irritating the vagi nerves in the neck*, infers that neither this stoppage, nor dilatation of the pupil, nor contraction of the vessels, are caused by galvanizing these nerves.—Proceedings Royal Society, May 12, 1871, p. 341.

[†] Union Médicale, 1835.

B.—HABITUAL FEEBLE ACTION.

329. Persistent impairment in the vigour of the heart's contractions attends a variety of organic changes of its texture, acute and chronic. But such impairment is also observable in some cases, where no appreciable failure or perversion of cardiac nutrition can be detected; and to these only is reference intended in the present place.

330. *Symptoms.*—Where the heart is habitually feeble in action, coldness and clamminess of the extremities, œdema of the ancles and insteps, shortness of breath, frequent inclination to faintness, sensations of languor and ennui, low spirits, anorexia or depraved appetite, foul breath, and constipated bowels, are more or less constant symptoms.

331. *Physical signs.*—The heart's impulse is feeble; its sounds want tone. Reduplication of the diastolic sound at the base is common, and palpitation easily excited.

332. *Treatment.*—This state of things, which may be principally seen in young females, and often in connection with disordered menstruation, is curable by attention to the state of the uterus, and by the tonic invigorating plan of treatment. Moderate walking exercise is essential, though commonly very much disliked by the patient. Horse exercise proves serviceable also.

V. ANGINA PECTORIS.

333. By the terms angina pectoris, syncope anginosa, and suffocative breast-pang, is understood a paroxysmal neurosis, in which the heart is essentially concerned. As far as it goes, this definition is, doubtless, unassailable; its logical defect is inadequacy; —a defect that cannot be got rid of, until our notions of the intimate nature of the affection acquire much greater solidity, than they at present possess.

334. *Symptoms.*—The dominant symptom of angina is pain; without this, the complaint cannot be said to exist. Next in

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* Cyclopædia of Surgery, art. Emphysema, p. 79, 1842. In a written communication from Dr. Stokes, the writer mentions that a suicide by forcible distension of the lung, through holding the breath, had just occurred in Dublin (1871).

importance, if not in truth equally essential to our idea of the affection, stands a sense of impending and speedy dissolution, preceded or not by faintness.

(*a*) The essential *seat* of the pain, in the great majority of cases, corresponds to the præcordial part of the lower sternal region,—as far as mere topography goes, it seems, consequently more directly connected with the right than the left side of the heart. By some exceptional persons, the pain is especially referred to the middle, or even upper, sternal region.

Associated with this pain, appears tenderness in the spot,—cutaneous, or at least superficial, *hyperæsthesia*. There is no positive evidence of increased tactile sensibility of the heart itself.

In *character*, the pain is rarely dull or aching; much more commonly gnawing, tearing, lancinating, cramp-like, or indescribable,—an exquisite torture, grasping, constrictive, and suffocative.

(*b*) Synchronously with the cardiac seizure, or a little later than this, there may, or may not, occur (the affirmative by far the more commonly) various painful sensations in other parts. Pain shooting to the mid-dorsal spine, to the left side only, or to both sides, of the neck and occiput, the left shoulder, the entire length of the left arm, or stopping short either at the insertion of the deltoid, or at the bend of the elbow, or limited to the middle inner aspect of that arm, or similarly affecting both arms, or much more rarely the right arm alone, or sometimes passing to the left leg, sometimes invading the four extremities at once, and occasionally involving even the testes,—pain thus localised, possesses the shooting, stinging, tingling, and benumbing qualities of neuralgia. These pains are habitually regarded as secondary, reflex, or sympathetic offsets from the cardiac nerves primarily involved.

(*c*) *State of the Pulse.*—Nothing can be more contradictory than the statements of writers concerning the state of the pulse in angina; cases free from *obvious* organic disease of the substance and of the valves of the heart have evidently not been discriminated from others in which more or less grave lesions of the kind existed. It is, further, very necessary to separate the pulse of the seizure from that of the interval.

Now the pulse in the interval, as far as I have seen, possesses no special character; in frequency and in force, and in rhythm, its attributes are those of health,—provided the disease be either ostensibly dynamic, or there be no obvious structural change present, capable of accounting for perversion in one or more of those characters. I have very rarely seen a patient in the actual height of an anginal paroxysm; but that towards its close, in cases of recovery, there most certainly need be neither acceleration nor irregularity of beat, I can aver from my own experience. Angina resembles epilepsy, in that the actual fit is relatively seldom witnessed by the medical observer; still cases have been recorded showing that in the very extremity of the pain the pulse may be perfectly regular, and scarcely exceed by half a dozen beats per minute the rate normal in the individual,—an increase of fifteen to twenty beats has, on the other hand, been observed.

In fits destined to terminate fatally, the pulse becomes almost imperceptible for some moments before death, and may or may not be disturbed in rhythm.

(*d*) *State of the Respiration.*—Dyspnœa, subjective or objective, is not an essential phenomenon of the anginal fit. The number of respirations per minute may *secondarily* be augmented,—though never to any notable degree; but a real sense of dyspnœa—a marked increase in the craving for breath—does not occur in the pure disease. Special hyperæsthesia of the medulla oblongata or pulmonary branches of the vagus is not, in other words, excited. It may, on the contrary, almost be affirmed that the special sensibility of the pulmonary nerves is temporarily maintained below par; for the *amount* of expansion with each inspiration is lessened, by combined consensualism and volition, from the dread of increasing the post-sternal pain, while the *number* of breathing acts in the minute scarcely undergoes any, and certainly not a commensurate, increase. By an effort of the will, if the patient have the courage to risk the chance of a moment's extra agony, the chest may be freely expanded. Nay more, there are exceptional cases wherein, as long since noticed by Parry, momentary release from suffering is obtained by taking a full inspiration, throwing the shoulders back, straightening the spine, and keeping at the "top of the breath."

I have known more than one anginal patient refuse to sit down during a protracted examination, from the fear of bringing on a paroxysm of pain. Some short while since a sufferer of the kind, sitting before me, suddenly started to his feet with a look of the most agonized fear: warned by "uneasiness" in the cardiac region, that a fit was imminent, he succeeded in arresting it by assuming the erect posture.

It may safely be assumed that in cases of angina, where marked lividity of the face and genuine dyspnœa exist, either the orifices or walls of the heart are gravely unsound, or pulmonary disease of some kind complicates the seizure.

(*e*) *Alimentary Canal.*—The gastric branches of the vagus are not always implicated. But violent and continued eructation (not explicable by the nature or recency of the last meals) and sometimes vomiting, every now and then prove the existence of irritation in those filaments. Flatulent distension of the entire abdomen is sometimes observed. There seems to me little reason to doubt that gas is actually secreted in some of these cases.

(*f*) *Nervous Centres.*—The mind habitually remains clear, the patient being conscious of all that goes on around him. The faculty of perceiving pain remains almost to the last moment vividly acute. There are instances in which the extreme anguish seems to deaden perceptive power in regard of all but itself; and patients on recovery will prove unable to remember any circumstances attending their past paroxysm but the pain itself.

Slight convulsions occur occasionally; the gravest tetanic spasms, with complete opisthotonos, have been witnessed. When the seizure has been of this kind, tonic spasm may hold on, in some spots, hours after the actual fit has passed away.*

(*g*) *Organs of Sense.*—In extreme cases, and not alone where death is near at hand, the sight may fail more or less completely.

(*h*) There may be a full discharge of watery urine.

(*i*) *General Symptoms.*—The general symptoms are those of nervous shock,—pallor of surface, cold clammy perspiration, fall in temperature of the surface generally, intense anxiety of countenance, and not only dread of approaching dissolution, but profound conviction that life cannot go on unless instantaneous relief be afforded.

335. *Physical Signs.*—The physical signs connected with the heart, provided no obvious organic disease exist, are not necessarily abnormal. The impulse and the sounds may be natural,—there is no murmur. If there be notable disturbance in any of these points, that disturbance is the exponent of pre-existent organic disease. Even rhythm is not of necessity disordered,—except where such disease is present. All this is *à fortiori* true in the intervals of seizures: a past attack of angina leaves no impress on the structure or actions of the organ.

336. *Mode of Seizure.*—The seizure, in the majority of patients perfectly sudden, occurs without the slightest warning; in rare

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* Allingham, U. C. H., Females, vol. xvii., p. 57.

instances the habitual sufferer receives notice of the coming fit through certain cardiac paræsthesiæ. He knows also that particular movements and acts will either positively, or nearly so, produce an attack.

I have recently seen a case in which the pain followed the reverse of the habitual course;—originating at the left wrist, and extending thence almost immediately to the heart, the paroxysm then became complete. Here the brachial pain played the part of an *aura*.

Seizures occur when: (*a*) the patient is walking quietly on level ground, up hill, against a strong wind, especially when these movements are undertaken shortly after a meal, and oftenest, as far as I chance to have seen, after the earliest meal in the day; or (*b*) when he is sitting quietly and undisturbedly; or (*c*), when in bed, either awake or, more frequently, asleep. In five fatal cases observed by myself, the patient died in bed; in three, awoke to die.

Emotional excitement is not so frequent a precursor of the fit as might be imagined. Such excitement may, however, precede a fatal paroxysm, even (as I have actually known) of quietly pleasurable character.

337. *Terminations and Duration of the Fit.*—Lastly, a few seconds or a few minutes, the anginal seizure may continue to recur, with variable intensity, several times during an hour or more; a paroxysm may thus be single or composed of a number of successive seizures. In either case, if the issue be favourable, it goes as suddenly as it came,—leaving as its sequence a feeling of general exhaustion, accompanied with a strong sense of present relief. Or death takes place in the manners to be presently described.

The termination of the attack is sometimes followed by temporary hydruria.

338. *Manner of Death.*—The immediate mechanism of death appears, as far as evidence goes, to be syncopal. But the conditions, directly antecedent, vary. Thus the fatal syncope may be perfectly sudden, instantaneous, coeval with a single pang,—there may be no time for struggle, for pulmonary congestion, for external convulsion: the most perfect placidity of expression and calmness of attitude are under those circumstances found in the corpse.* Or death may be a more deliberate process: the pulse

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* In a case of this species, where death occurred instantaneously while the patient was reading quietly in bed, there was so little disturbance of the frame, that his thumb

gradually fails; fits of brief duration follow each other rapidly; anxiety and dread of dissolution reach their possible limit; the eyes start from the orbits; cold clammy perspiration breaks out on the surface; the radial pulse becomes imperceptible; the breathing grows laboured; trismus with clonic or tonic spasms of the limbs, or it may be of the entire trunk, occurs; insensibility follows, and in this state the sufferer rapidly perishes.

339. *Recurrence.*—Angina belongs essentially to the class of recurrent affections,—the fact of one seizure having taken place is a reason why others should follow. And with each returning seizure the activity of the directly exciting cause is generally noticed to have lessened; so susceptible does the nervous plexus concerned become, that an influence which at first would have failed to excite, eventually readily produces, a paroxysm.* The first seizure, often, as we have seen, comes of effort of some kind,—more particularly that of walking briskly up a hill or even on level ground. Sauvages (1763), exaggerating the importance of this character, makes its occurrence a *sine quâ non*, by giving it a prominent place in his definition of the disease. The patient, forced by the agony to stop, is at once released through the stoppage from his suffering, steps forward again, is again brought to a standstill; and this process having been repeated a variable number of times, walking becomes possible,—it may be even with enjoyment; or, either from dread of the pain, or from real return of it every time the effort is renewed, the attempt is by-and-by relinquished.†

Now, a paroxysm of this kind may, it is affirmed, remain single for a lifetime of ordinary duration, provided all conceivable exciting causes be carefully avoided. I have known instantaneous death occur from angina in a male, aged about forty-five, who had but three fits,—the first one year, the second half an hour, before the final and fatal one.

But such distant recurrence is not the rule,—the intervals of freedom gradually or rapidly shorten; and eventually emotion of any kind, sudden movement, the acts of defecation, of coughing, of quickly swallowing water, will induce a fit; or no appreciable cause of any kind may precede. Attacks occasionally, but cer-

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and forefinger were found in the pamphlet on which he had been engaged; the bedclothes, too, were undisturbed (Case seen with G. Bird.)

* See, on the general question of "Recurrence," "Lancet," vol. i., 1871.

† "I can walk with ease ten or fifteen miles" (said a patient to me, *within three days of his death in a paroxysm*), "after I have been stopped three or four times at intervals of a hundred yards." This person succeeded well in walking up hill *zigzag and sidewise.*

tainly not commonly, take place as the patient drops off to sleep. It is said the returns are sometimes distinctly periodical,—and that this is one of the numerous forms malarial neurosis may assume.

340. *Duration of the Disease.*—In regard of this point the greatest diversity prevails.

On the one hand life may be prolonged for years after the first seizure, and in spite of more or less frequent recurrence. I have met with an instance in which there was the strongest evidence that the first paroxysm had occurred *twenty-four years* prior to my interview with the patient.

At the other extreme, as matter of recorded fact, must be placed a case published by Latham, where death took place in about two hours after the first and original outbreak of pain. Now this case justifies us in supposing that the total duration may be yet briefer,—and that, occasionally, instantaneous and unwitnessed death, occurring in persons previously believed to be healthy, has been accompanied with the phenomena of angina. Be this as it will, there is no doubt that in the generality of instances the complaint runs a somewhat protracted course. Data are wanting wherefrom to calculate the mean duration; did we possess the information, it would be practically inapplicable to individuals, and prove simply a contribution to the natural history of the disease.

341. *Anatomical conditions.*—First there are few, if any, structural diseases either of the heart, its orifices, and its nutrient arteries, or of the aorta, which are not to be found recorded in the narratives of the *post-mortem* examinations of different victims of angina pectoris. Secondly, there is no conceivable disease of those structures and parts which has not in various individuals reached the highest point of development, without anginal paroxysms, even of a slight kind, having occurred during life: to this proposition extensive calcification of the coronary arteries perhaps furnishes a solitary exception. Thirdly, the organic changes most frequently met with have been fatty atrophy and flabby dilatation of the heart; obstructive disease of the coronary arteries by atheroma and calcification; and *calcification of the arch and orifice of the aorta*, more or less markedly interfering in turn with the orifices of the coronary arteries. Fourthly, the rarest changes have been hypertrophy and hypertrophy with dilatation. In truth, it may be doubted whether these conditions in their genuine form, without any combination of fatty metamorphosis, have ever been the sole morbid states present.

Now the conclusions from these premisses cannot be other than that, even in the most serious case of textural change, there is neurotic disturbance beyond that change as the essential element of the paroxysm;—that textural change acts simply as a promoter of the seizure by rendering the nerves in some way more apt to suffer dynamic disturbance;—and that the forms of organic disease most favourable to that disturbance, all of them, indicate mal-nutrition and weakened power in the parts concerned.

If we must place implicit faith in all recorded observations, we are called on to admit that death may be produced by angina, the heart and vessels being texturally sound. I confess I cannot help doubting this. Recent narratives invariably describe some organic change; and older accounts are not trustworthy, seeing that accurate knowledge concerning dilatation, and various forms of mal-nutrition has been the acquisition of the last few years only. My own experience, as far as it goes, emphatically deposes to the constancy of structural disease. It has occurred to me to examine during life some twenty-four cases of thoroughly well-characterised angina; in every one there were physical signs of organic change, either in the heart, the aorta, or both. The signs and symptoms indicated: (*a*) In the case of *the heart's* substance, either flaccid, flabby enlargement, associated or not with sub-pericardial fat or fatty metamorphosis,—never pure, sthenic hypertrophy, though the character of the impulse and of the arterial pulse occasionally made it very probable an excess of feeble muscular fibre had formed. (*b*) In the case of *the valves*, aortic stenosis, very, very rarely insufficiency, and never, as far as I can recall, mitral disease, either stenotic or reflux. If the latter experience be also that of observers in general, the fact must be regarded as a very important one in the natural history of the disease. (*c*) In the case of the aortic arch, widening, saccular or not, with calcification. I have, besides, opened, or seen opened, the bodies of five persons destroyed in the actual paroxysm; the heart or aorta, or both, were texturally affected in all.

But it is accordant with my experience to add that fatal angina is more to be dreaded in association with organic defects, either difficult or impossible to diagnose (such as slight fatty metamorphosis and calcified coronary arteries), than with those grave forms of structural mischief that are readily discoverable by physical examination.

Has angina pectoris, then, any *anatomical character?* In all the cases I have opened there was more or less (sometimes, however, very slight) calcification with contraction of the coronary arteries; and my memory (which may, however, be treacherous) does not furnish me with any example of very highly developed and extensive calcification of those vessels where anginal symptoms had not existed in more or less definite form during life. Still, calcification of the coronary arteries has not always been found by other observers [346].

342. *Appearances dependent on Manner of Death.*—The appearances dependent on the manner of death are among those of syncope. Here were, for instance, the essential conditions discovered in the body of a male, aged sixty, cut off in the paroxysm:—

Twenty-six hours after death: weather cold, damp; *cadaveric rigidity extreme;* vibices posteriorly abundant; subcutaneous thick layer of fat; face pale, without lividity; no fluid in either pleural or pericardial sacs; old pleural adhesions posteriorly; dark congestion, moderate anteriorly, both lungs; large mass of fat attached to edge of base of right lung; much sub-pericardial fat; *heart very flabby, may be rolled up,* not notably enlarged as a whole; walls neither specially thick nor thin, except about right apex, where the sub-pericardial fat has encroached on the muscular substance; *a little fluid blood in four cavities, no coagula; some fluid blood in pulmonary arteries,—neither thrombus nor embolus;* no valvular disease of any kind; *very moderate atheroma and calcification of coronary arteries; aorta, opened in entire length, calcified extensively, with minute superficial fissures here and there. Very partial and incipient fatty metamorphosis of heart's fibre.*

The total absence of firmness, and of any indication of spasmodic contraction,* of the heart (while cadaveric rigidity existed in the external muscles to a very marked amount) is worthy of note,—also the almost complete absence of blood from the heart's cavities, and the freedom of the pulmonary vessels from thrombal or embolic obstruction.

343. *Predisposing causes.*—Angina predominates vastly in males; the ratio of ten to one is deducible from some published estimates. In all probability the excess in males is in considerable measure explicable by the greater frequency, with which they suffer from disease of the valves and mouth of the aorta.

The disease is rare before the fiftieth, excessively so before the fortieth, year,†—and unknown in infancy and childhood.

Angina is certainly very materially more common in the better than in the humbler ranks of society; and my own experience, confirming that of Gilbert Blane,* scarcely supplies me with more than a solitary well-defined example of true angina in hospital practice.

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* In all cases I have seen after death, the heart's fibre was similarly flaccid.

† Of 88 cases, collected by Forbes, 80 refer to males; and of 84 patients, 72 had passed the fiftieth year.

I have seen a case or two leading me to surmise that spanæmia, where other conditions are favourable, will promote the occurrence of seizures.

Laennec supposed that epidemic influence renders the disease unusually common from time to time.

It seems doubtful what part (if any) gout may play in facilitating the occurrence of true angina: the disease often occurs of necessity in gouty people, because gouty, like anginal, sufferers are habitually elderly males of the well-to-do classes.

The abuse of tobacco has appeared to me in some instances to have promoted the development of the disease.

344. *Diagnosis.*—Whether an intra-thoracic pain is really anginal proves, as a general rule, not difficult of determination. The intensity and locality of the suffering, and attendant dread of impending death, suffice commonly to distinguish it. Still the affection is occasionally simulated more or less closely by some one of the conditions grouped together further on under the heading of Pseudo-Angina,—to the description of which the reader is referred for the means of distinction.

Admitting a given case to be one of angina, it remains to ascertain the structural conditions of the heart. The presence or absence of far the greater number of organic diseases can easily be determined by the rules given with the account of each of the series. But it unfortunately happens that the two organic affections very commonly associated with angina, namely, fatty atrophy of the muscular substance, and calcification of the coronary arteries, are precisely those least susceptible of sure diagnosis. Demonstration of the latter condition lies beyond the limits of existing knowledge wholly; and of the great difficulty attending the detection of the former, unless in its very aggravated forms, enough is said with the account of the disease.

345. *Prognosis.*—The life of him who has had an unmistakeable attack of angina is not insurably safe for an hour. And yet existence *may* be protracted for years. Is there any method whereby we can with a reasonable amount of security place a given sufferer among those who shall be cut off perhaps the next instant, or among those who shall survive for years? I know of none. The presence or absence of demonstrable organic disease

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* Med. Chir. Trans., vol. iv.

and the quality of this, so far from guiding us correctly, may prove the source of complete error. I have examined a man in whom nothing physical could be detected except feebleness, by no means great, in the cardiac action, who perished forty-eight hours later in the paroxysm,—*post-mortem* examination proving the absence of any structural change except moderate softness of fibre, with a very slight, almost problematical, amount of intra-sarcolemmous fat. And *per contra*, it has occurred to me to treat a certain number of cases, where, in spite of different forms of obvious organic mischief, life has held on for months, though paroxysms have all the while more or less frequently recurred. Hence in my mind there is great fallacy in the doctrine that so-called "functional" angina ("functional" doubtless solely because textural change eludes detection) is a relatively safe complaint, and "organic" angina alone pregnant with constant danger. Nor will the mildness of an attack, actually witnessed by a medical observer, give warranty for his uttering a reassuring prognosis: the next attack within an hour or two (as I have actually known) may destroy life.

In short, the *cardinal fact in the prognosis of real angina under any circumstances is its uncertainty.*

346. *Nature, seat, and mechanism.*—That angina pectoris is in its essence neurotic, appears from the sudden advent and departure of the seizures,—from the character of the attacks while actually present,—from the perfect ease enjoyed in their intervals,—from the impossibility of surely assigning to the affection any special anatomical character,—and from the kind of treatment that proves beneficial.

Now pain is obviously enough the essential neurotic element of the disease. As to the nerves involved, the statements made concerning cardiac neuralgia seem distinctly applicable [254]. Plainly the pain has nothing to do with the chest-wall,—it is neither cutaneous nor intercostal: it is at the least intrathoracic; *and the final influences it exercises on the functions of the heart in fatal cases, as well as the manner of its radiations,*[*] *localise it in the cardiac plexus.*[†] The special proneness of these nerves to suffer arises probably in all, certainly in the great majority of cases, through the disturbing influence of structural changes in the heart.

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[*] Vide Mueller's Physiology, by Baly, vol. i. p. 666.

[†] Since the last edition of this work was published a most satisfactory proof that the view then put forward, and now reproduced, is sometimes, at the least, correct, has been given by Lancereaux. This observer found in a fatal cases of angina vascularity, with exudation, of the portion of cardiac plexus lying on the aorta between the greatly narrowed coronary orifices. There was a condition of the aorta in the spot that might be called *cirrhosed.* (Gazette Méd. de Paris, p. 432, July, 1864.)

The mechanism of the neurosis seems centric (encephalic), reflex, or intrinsic, according to circumstances. We can scarcely doubt centric (encephalic) origin, when the paroxysm is excited by anger or other strong emotion:* when it follows the ingestion of a full meal, or the blowing of wind on the face, we can hardly question the reflex agency of the gastric vagus and the trifacial: when the agony instantly succeeds effort or abrupt movement, the direct influence of the circulation through the heart's texture on its own nerves seems clearly indicated.

But with this pain, its essential element, does any other neurotic disturbance co-arise in the paroxysm? Heberden and Latham espouse the doctrine of cardiac spasm,—but whether this spasm be the result of the pain, or, as ably argued by the latter observer, its cause, has been admitted to be an unsettled point. Parry and Stokes, on the contrary, appealing to clinical observation, assume temporary increase of weakness in an already weakened organ, as the essential muscular element of the anginal paroxysm.

For my own part I regard the presence of spasm, either initiative or sequential, either simple or with cramp [277], as altogether doubtful in the *ordinary* class of cases. In the first place it surely appears incontestable that anginal suffering may exist, and in a well-developed form, without spasm. For how is it possible that a heart spasmodically contracted can act rhythmically, as all close observers admit the organ habitually does, where no *organic* disease, of necessity influencing the healthful measure of the pulse, co-exists? And if the seizure may thus be pushed to the imminence of death without spasm, it seems unlikely the mode of action should change just at the moment of the fatal syncope.

I cannot help believing it most probable that the pain, when it gravely affects the motor innervation of the heart, does so paralytically—that the death is one of suspended and not perversely excited contraction. When the pulse does suffer change, its dominant condition is increasing weakness; besides, in the few cases I have opened, where the patient had been cut off in the paroxysm, the heart bore *no marks of recent spasm*,—it was flaccid and yielding. Further, who ever meets with genuine angina in the possessor of a strong purely hypertrophous heart? And, again, where death does appear to result from cardiac spasm, as in poisoning by digitalis [p. 174], the conditions, preluding death, are widely different from those of anginal dissolution.

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* We may accept as granted that the immortal Bichat erred in the doctrine he so firmly espoused, as to the *primary* and *direct* influence of the emotions on the nerves of the heart, to the exclusion of any portion of the great nervous centres.

But, while holding on these grounds to the doctrine, that weakened force, culminating in paralysis, is truly the perverted motor state in *ordinary* angina, I am not unprepared to believe exceptional cases may arise in which simple spasm, or spasm with dislocation or rupture of fibre (true cramp), really occurs. It seems possible, at least, that in those rare instances where intense tetanic spasm invades the external muscles, an analogous state of the heart may be the real cause of its stoppage; but I should not, even so, be disposed with Latham to look on the pain as the effect of the spasm, but to view the spasm as the reflex disordered motor result of the pain. At the same time it must be admitted that no such rupture has ever yet been positively seen. Even microscopical examinations failed to detect any such condition in the cases I have examined; let me add, however, they were clearly paralytic cases as far as I could judge.

It is not unworthy note how excellently the phenomena of angina occasionally contribute to demonstrate the independence of the nervous forces severally exciting mere muscular contraction and rhythmical motion of the heart: while the former is gravely and increasingly affected, the latter may maintain the even measure of health almost to the last moment of existence. Pathology might, here, as in so many other instances, have served as a guide to physiology.

347. *Treatment.* (*a*) *During the fit.*—Sedatives and stimulant anti-spasmodics are the medicines essentially to be trusted to during the fit. The dose of opium will be measured by the intensity of the pain; from forty to sixty or eighty drops of laudanum or of the liquor opii sedativus may, in a severe case, be given along with brandy, or from half a drachm to a drachm of æther or aromatic spirits of ammonia, and repeated according to the urgency of the suffering. Musk, camphor, and belladonna are of very inferior importance. Mustard poultices may at the same time be applied to the heart and to the dorsal spine, or cloths wetted with the strong liquor of ammonia laid upon the præcordial surface. Laennec's suggestion of the transmission of a magnetic current (with or without acupuncturation) through the chest, has scarcely been fairly tested. An electro-galvanic current, however, affords

better chance of successful influence, and in a serious case of quickly recurring paroxysms, which seemed indisposed to yield to the ordinary sedative management, deserves trial. Of the necessity of cautious proceeding in this trial enough has elsewhere been said [328, *c*]. The mustard pediluvium, especially if the patient be gouty, is useful; the application of chloroform to the præcordial region, hazardous. Inhalation of small quantities of æther, chloroform, and especially of nitrite of amyl,* have been strongly recommended: none of these agents (though their occasional good effects are strongly testified to) can be employed without a certain amount of risk. Hypodermic injection of morphia, as also of atropia, has occasionally proved more efficacious than internal opiates. Large doses of chloral are certainly very dangerous.

Speciality in the circumstances of the attack may call for special treatment.

If the patient be the subject of undoubted sthenic plethora, and especially if the heart be known by previous examinations to be, contrary to the rule, a well-nourished one, the abstraction of blood from a vein, or by cupping between the shoulder-blades, has been held to be clearly indicated; but bleeding must not be heedlessly undertaken, and without assurance, as positive as is attainable, that the heart is at least not a dilated, soft, or flabby one. It has not occurred to me to meet with a case in which bleeding seemed warrantable.

If flatulence and acidity exist, soda, cajuput oil, and sesquicarbonate of ammonia may be administered with the opiate medicines; if a large undigested meal lie in the stomach, it should at once be removed by an emetic of sulphate of zinc.

(*b*) *Prevention of fits.*—A paroxysm, of which a small minority of experienced patients learn by their feelings to expect the approach, may sometimes be averted completely by an opiate; and sufferers should always carry, properly protected, on their persons, an antispasmodic and sedative draught. One of the best safeguards against seizure consists in the idea, that the means of averting evil consequences, if it occur, are within reach. In the rare instances, where an *aura* from the left forearm ushers in the fit, this might possibly be prevented by a ligature applied above the elbow.

(*c*) *In the intervals.*—A person who has had one attack of angina pectoris must remember, that instances, in which recurrence does not take place, are altogether exceptional, and further, that the periods of recurrence gradually approximate more and more, while each successive paroxysm, as a rule, exceeds its predecessor in severity. If it be true, as we have seen, that a first attack is often brought on by an effort of some kind, such as walking up a hill, or in the teeth of a sharp wind, on the other hand, the most trivial influence will eventually suffice to produce a paroxysm; emotion of any kind, sudden movements of the trunk or arms, efforts at defecation, the acts of coughing, drinking rapidly, &c. Hence, it is clear, the subject of angina must live according to the most stringent rules; every conceivable precaution must be taken to keep the heart in a tranquil state. The patient should give up exciting pursuits of all kinds,—intellectual, corporeal, and emotional,—and learn, if he be of the *genus irritabile*, to govern his temper. Daily exercise should be slowly taken on perfectly level ground, either on foot or in an easy carriage; riding on horseback is scarcely to be permitted with safety. The diet should be moderate in quantity, simple in quality; the bowels never allowed to be confined. A belladonna plaster worn over the heart, and an issue, seton, or perpetual blister to the arm, have appeared useful in some cases; if the patient have confidence in counter-irritation, this should by no means be neglected. Change of scene and travel, coupled with the use of tonics, vegetable or mineral, will, by improving the general health, render the patient (as a matter of reasonable probability, but not of certainty) less prone to seizures. Arsenic, nitrate of silver, and sulphate of zinc are the best of the class of mineral tonics, unless anæmia be present, when, of course, iron is *the* remedy. The removal of gout, in its genuine arthritic form, of chronic rheumatism, or old-standing skin-diseases should be very cautiously, if at all, attempted in the subject of angina: relief of those complaints is unquestionably sometimes followed by increased severity of the cardiac affection.

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* Dobell's Reports, vol. ii. p. 441; also Wilkes, "Lancet," 1869, vol. i. p. 85.

In a case having all the attributes of true angina, the attacks, which were frequent, ceased under the influence of *small* doses of chloral (ten grains) taken twice daily.

If angina of malarious origin and periodical recurrence really exist, its treatment would be that of miasmatic diseases generally.

VI.—Pseudo-Angina Pectoris.

348. Genuine angina pectoris is undoubtedly a very rare affection. On the other hand, I *almost daily* meet with a form of complaint combining in a minor degree many of the characters of

angina; and to this imitation of the true disease I propose to give the name of pseudo-angina. I believe that herein lies the explanation of Laennec's notion (so discordant with the experience of English observers), that angina pectoris is of very frequent occurrence.

349. *Symptoms.*—The symptoms of pseudo-angina are more or less severe, commonly constrictive, pain referred to the region of the heart, and palpitation occurring paroxysmally without obvious cause, or under exertion, or through over-eating, indigestion, flatulent distention of the stomach, and a variety of other functional disturbances. The breathing becomes panting, and suspirious. Giddiness and faintness are sometimes observed. The patient dreads efforts of all kinds.

350. *Physical signs.*—Physically the heart may be to all seeming healthy. The apex beats in the usual spot, and with natural characters; even in the height of the paroxysm no murmur of any kind exists. Shortness, feebleness, and heightened pitch of the first sound at the apex are the only conditions at all frequently met with. These indicate tendency to dilatation, and weakened power: the impulse sometimes wavers, and is of uneven force from time to time. But even in cases, where all seems physically normal, there may be organic disease beyond the diagnostic means of the present day: if so, it must certainly be slight.

351. *Predisposing causes.*—Like true angina its imitation is more common, though by no means to the same extent, in males than females, and in the higher than in the lower walks of society: but, unlike true angina, the pseudo-disease frequently occurs in young adults.

352. *Associated conditions.*—Pseudo-angina has fallen under my notice as an attendant on various hæmic diseases,—chronic gout and rheumatism, and spanæmia: on nervous diseases,—hysteria, spinal irritation, and epilepsy; on intercostal and mammary neuralgia, the direct evidences of which are more or less complete.

Pseudo-angina also occurs in connection with a variety of nervous disturbances, the influence of which can only be made intelligible by reference to some physiological facts. That *suddenly* perverted dynamism of the brain and cord affects the heart's action is proved by the phenomena of simple concussion of those centres; while the like influence exercised by irritation of the roots of the spinal accessory, of the first four cervical nerves, of the par vagum, of the phrenic and some intercostals, of the cervical ganglia, especially the first, of the cardiac nerve, of the semilunar ganglia and solar plexuses, of the nerves of the stomach, of the hepatic, ovarian, uterine, and even cutaneous nerves, has, in each instance, been proved by more or less precise experimentation. The physiological sympathies of the heart being thus extended, it is not to be wondered that functional disturbances of almost every organ may entail derangement of cardiac action and feeling. Of feeling, for there seems fair reason to suppose that the heart, though unendowed with tactile sensibility in health, may acquire it in disease and under the influence of special impressions [260].

353. *Diagnosis.*—Hysterical and anæmic palpitation, accompanied with intercostal neuralgia, nay, even organic palpitation in a person with that form of neuralgia,* may readily be mistaken for true angina; indeed, an attempt has been made by some French writers to show that the latter disease is nothing more than a "brachio-thoracic neuralgia," the heart-symptoms being purely accidental. In true angina the points of tenderness in the course of an intercostal nerve are wanting; besides, the severity of the suffering in the cardiac region is infinitely greater than in intercostal neuralgia.

I some time since had, under occasional observation, a singular case of a youth who had periodical returns of severe neuralgic pain *originating* at the insertion of both, but especially the left, deltoid muscles: præcordial pain, with slight or grave syncopal feelings, *following* [336]. There was dilated hypertrophy, associated with systolic basic murmur, and certain anomalous physical phenomena. But the functional state was not truly anginal.

354. *Treatment.*—Although common sense and experience point to the necessity of learning the cause of pseudo-angina, in order to cure the effect, still, in instances where we either fail to discover that cause, or find it to be itself non-removable, much good may be done to the cardiac symptoms by direct treatment of these on the principles applicable in cases of true angina.

And this is the more true, inasmuch as possibly no *absolute* symptomatic distinction may exist, after all, between the *true* and the *false* forms: it may be that they represent the *gravior* and the *mitior* epilepsy,—the *haut* and the *petit mal:* but that there is radical distinction in the *primum mobile* of the seizures no doubt can be entertained: in the false disease there is no disease of the aorta or of the cardiac plexus.

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* Roberts, U. C. H., Females, vol. v. p. 217; Hawkesford, id., vol. v. p. 1.

SECTION II.—ORGANIC DISEASES OF THE HEART.

§ I.—ANÆMIA OF THE HEART.

355. Although anæmia of the texture of the heart has not attracted notice on the part of systematic writers, there can be no doubt of its real existence as a definite morbid state. However, as actually seen, it commonly forms an element of some complicated form of change, to which attention is, excusably enough, perhaps, mainly turned.

356. Anæmia gives a mottled pallor to the cardiac substance; and, if the conditions inducing it have been of long standing, lessens the amount of muscular tonicity and firmness. The softness and pallor accompanying obstruction of the coronary arteries, and the early period of fatty atrophy of the fibre, are examples in point. In extreme general spanæmia the pallor of the heart is striking. Now it is well known that such pure unassisted spanæmia occasionally destroys life; and the difficulty of accounting for the fatal event has been recognised by Chomel and others. In a case of the kind, where very careful examination, both before and after decease, failed to discover any commonly acknowledged cause of death, I was struck with the intense pallor of the heart, —and it appears not unreasonable to admit that the slow extinction of the patient, by a sort of protracted syncope, may have been chiefly effected through the gradually lessening contractility of the heart's fibre, fed as this was by imperfect blood.* In the case referred to, there was no leucohæmia or enlargement of the spleen; nor were the supra-renal capsules diseased, or the skin discoloured.

357. In cases where anæmia has either been persistently, or with occasional intermissions, sustained for a more or less lengthened period, it is probable changes are effected in the structure and properties of tissue of the heart. The weakened power of resistance of the ill-nourished muscular substance must, it can scarcely be questioned, facilitate the occurrence of dilatation of the heart's cavities, where efficient extrinsic causes of that dilatation are at work. On the other hand, hæmorrhagic spanæmia, even of the gravest kind, will not reduce the resistent force of the walls of the ventricles so much as, unassisted, to lead to such dilatation: the proof of this is, that the hearts of persons cut off by cancer

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* Monk, U. C. H., Males, vol. xii. p. 71.

of various organs, though highly anæmic, are not necessarily dilated.

§ II.—Congestion of the Heart.

358. The substance of the heart, and of its membranes, as of all organs, is subject to three species of congestion, active, passive, and mechanical. There can be no doubt these forms of congestion contribute their share, sometimes an important share, to the sum total of functional disturbance occurring in different cardiac diseases. Yet they have attracted little attention from clinical observers; because they are, at least commonly, lost *post mortem* in the graver anatomical changes with which they are associated, and because, during life, they lie without the pale of sure diagnosis either through subjective symptoms or objective signs.

I.—ACTIVE CONGESTION.

359. Active congestion forms of course an element of the different inflammations that affect the heart and its membranes. But there seems fair reason to believe that active congestion, at least of the pericardium, may occur independently of the sequential exudation-process. In certain cases of rheumatic fever and of "Bright's disease," I have known excited cardiac action, uneasy sensations in the præcordial region with tenderness under pressure, accompanied with slight but unmistakeable pericardial rub, pass off without any evidence of fluid effusion into the sac. Is it not reasonable to admit (as it is certain vascularity alone may cause friction-sound [131, 375, *b*]), that in such cases simple active congestion has occurred and passed away without issue?

And, again, cases now and then occur tending to show the reality of active hydropericardium; active congestion must, if such cases be real, lead the way, preceding the escape of serosity.

II.—PASSIVE CONGESTION.

360. Although it would appear probable the heart's circulation must tend to stagnate during the course of adynamic diseases, the evidence of such condition is wanting. In typhoid (Peyerian) fever, for instance, instead of being abnormally loaded with blood, the minute vessels of the heart seem to contain less fluid than natural. Louis insists on the dry look and deficiency of usual moisture on the surface of sections of its tissue.*

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* Affection Typhoïde, t. i., p. 330, ed. 1.

III.—MECHANICAL CONGESTION.

361. Mechanical congestion of the heart's texture and membranes is of very frequent occurrence.

362. *Anatomical characters.*—Slightly increased width of the walls of the ventricles, fulness of the great cardiac vein and of its branches and their radicals, undue moisture and deep vinous coloration of the muscular tissue, with minute dark-coloured ecchymoses under the pericardium and endocardium constitute the anatomical characters. The right cavities of the heart are commonly loaded with blood.

363. *Causes.*—Obstructed circulation through the lungs, direct pressure either on the large venous trunks supplying the right auricle or on the great cardiac vein, and obstruction to the onward movement of the blood through the cavities of the heart, are the essential causes of this form of congestion.

Hence it is an attendant on asphyxia, and on capillary bronchitis, especially when superadded to emphysema; is seen in all cases of mediastinal tumour and aortic aneurism, when pressure chances to bear on the vessels just named; accompanies cyanosis dependent on constriction of the pulmonary artery; adds to the mischief in tricuspid regurgitation and constriction; and may even eventually be induced in sequence to capillary stasis in the lungs, arising from disease of the mitral orifice.

364. *Diagnosis.*—I know of no means by which this state may be directly diagnosed; but its existence becomes matter of fair inference under the circumstances just mentioned.

365. *Effects.*—The effects entailed by mechanical congestion of the heart will depend on the abruptness or slowness with which it is produced, and on the brevity or length of its duration.

When suddenly affected, it seems possible, such blood-stasis may induce hydropericardium,—as I shall endeavour to show with the history of that affection.

When slowly effected and long maintained, congestion of the kind must interfere with the heart's nourishment. This interference, in turn, theoretically lessens the power of resisting the excentric pressure of the blood on the part of the walls of the organ; hence dilatation of the cavities follows. And as matter of observation dilatation is found with tolerable frequency in cases of pressure of veins by intra-thoracic tumours and aneurisms. True, in aortic aneurism, such dilatation may be wholly wanting, even where the sac springs from the immediate vicinity of the sygmoid valves; but may not this depend, in some cases at least, on accidental deficiency of the pressure in question?

And further, the probabilities are that long-continued stagnation will end in local exudation-process of low type,—the effused fibrine, converted into induration-matter, must gravely impair the contractility of the infiltrated parts, and so in another form lay the ground-work of dilatation.*

§ III.—Accumulation of Blood in the Heart's Cavities.

366. Accumulation of blood may occur in any, or in all, of the cavities, in cases of endocarditis, as a consequence of fibrinous particles interfering with the free play of the valves, and in cases of rupture of valves. Accumulation in the right cavities, especially, will ensue in prolonged fits of palpitation, with gravely disturbed rhythm; in cases of tricuspid regurgitation; during fits of dyspnœa, in highly marked emphysema of the lung, especially if this has already led to organic dilatation of the right cavities; in capillary asphyxiating bronchitis; in acute pneumonia; and, in a word, to a greater or less extent, in all cases of notably obstructed circulation through the lungs.

367. *Symptoms.*—The symptoms, associated with such loading of the right heart, are dyspnœa, even to orthopnœa, dry cough, venous congestion of the face and upper surfaces generally (unattended, unless there be prior anasarca of the lower extremities, with œdema), oppression, anxiety, and sometimes pseudo-anginal feelings.

368. *Physical signs.*—The heart's impulse, laboured and struggling, irregular in force and rhythm, is seen and felt more extensively than natural, especially to the right of the sternum, and at the epigastrium. The area of dulness exceeds that of health, especially about the right costal cartilages.

369. *Treatment.*—Venesection, cupping over the præcordial region, sharp, rapid purgation, counter-irritants to the lower limbs, or the application of Junod's apparatus, are the remedies theoretically indicated for the relief of this state; practically, too, they prove efficient. But the condition, on which the obstruction of the cavities depends, remains of course in the background, unmodified, or scarcely modified, by them. Ulterior measures must be taken for the removal of that condition, if possible.

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* W. Jenner, Med. Chir. Trans., vol. xliii.

In the foregoing brief description, I have solely had in view the effects of accumulation of *fluid* blood in the cavities; the phenomena observed, when the *clotting-process* takes place, are considered under the head of INTRA-CARDIAC THROMBOSIS AND EMBOLISM.

§ IV.—CARDIAC INFLAMMATIONS.

I.—PERICARDITIS.

370.—Pericarditis, or inflammation of the proper tissue of the pericardium, is clinically known in the acute and chronic forms.

A.—ACUTE PERICARDITIS.

371. *Anatomical characters.*—The anatomical stages of completely evolved acute pericarditis are five: those of dryness with vascularity; of plastic exudation; of liquid effusion; of absorption; and of adhesion.

(*a*) The dry stage is marked by florid, arborescent, and capilliform injection, prominent tufts of capillary vessels sometimes standing out visibly on the surface,* especially on the cardiac portion of the sac; the membrane itself is dry and parchment-like. This stage is very rarely seen except where persons, dying of other diseases, are seized with pericarditis, just before dissolution.

(*b*) In the exudation-stage plastic lymph of various degrees of firmness accumulates in one or other of the following situations: on the cardiac and parietal serous surfaces both; on the cardiac division of the membrane only,—never, as far as I happen to have seen, on the parietal portion solely; on the posterior aspect only of both cardiac and parietal portions; on the anterior surface of the right ventricle only; or specially about the roots of the great vessels. The lymph is laid down stratiformly, in thick or thin layers; or in lace-like fashion, resembling the intra-cranial osteophyte of pregnant women;† or in hilllocks; or, like the sand on the sea-beach, in regular layers separated by indentations; or locularly; or in loose thread-like manner. Three-quarters of an inch is the extreme thickness I have witnessed in a layer of lymph,—and this only about the great vessels. Essentially of straw-colour in itself, it is reddened by blood-vessels, developed *de novo* within itself, and by imbibition of blood.

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† F. Parker, U. C. H., Males, vol. iv., p. 177, Dec. 1848.

* Vide Products, Adventitious, Cyc. of Anat. and Phys., p. 135.

In a fresh attack of pericarditis, lymph will not be thrown out on the immediate surface of old "white patches," whether these be of inflammatory origin or not.* And pressure, acting on any particular portion of the pericardial surfaces, will not only prevent the exudation-process, but even active congestion, from occurring at the spot. The best example I have seen of this fact occurred in a case where hydropneumo-thorax on the left side played the part of the compressing agent.† This peculiarity is evidently suggestive in regard of the *local therapeutics of inflammation.*

(*c*) The liquid effusion of the third stage varies in composition and qualities. It may be sero-albuminous and flocculent; sero-fibrinous and spontaneously coagulable after removal from the sac; hæmorrhagic; sero-purulent,—pus in sufficient quantity to be recognised with the naked eye being very rare; or, lastly, it may be wholly composed of pure pus. The latter condition of the fluid is, however, so rare, as almost to take rank among the *mirabilia* of morbid anatomy.

The quantity of liquid commonly ranges between three or four to ten or twelve fluid ounces. But when the amount of plastic exudation is abundant, and when death has occurred very rapidly, only an ounce or two may be found between the meshes of that exudation. On the other hand, where the affection runs a lingering course, enormous accumulation may occur,—reaching, in one case which fell under my notice, sixty measured ounces.‡

The closed sacculi, probably glandular, described originally by Lower, and again recently by Corvisart, as seated at the base of the pericardial sac, have not been shown to exercise any definite influence on the effusion-process.

The liquid macerates, softens, rarefies, and so promotes the distension of, the sero-fibrous texture of the sac.

I have never seen actual ulceration or sphacelus of the serous tissue.

Pneumo-pericardium, resulting from chemical changes in the fluid and non-dependent on sloughing of tissue, appears to have fallen under the notice of observers in singularly rare instances.

(*d*) In the absorption-stage serous fluid is removed with ease and rapidity,—hæmorrhagic effusion with less facility, and pus with still greater difficulty. Exudation-matter of variable plasticity remains behind,—to form the material of adhesions, loose or agglutinative, and to furnish a nidus either for the reception of various saline or animalized precipitates, or for the development of sundry pseudo-tissues. In some cases even exudation-matter itself appears to be partially removed by absorption [379].

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* Truman, U. C. H., Females, April 5, 1859.

† Plympton, U. C. H., Males, vol. iv., or Clin. Lectures, Lancet, 1849, vol. i., p. 579.

‡ Bartlett, U. C. H., Males, vol. iv., p. 292.

(*e*) In the final, or adhesive, stage the two pericardial surfaces may become universally agglutinated together; or locally adherent, either closely by patches or loosely by bands; lateral union of the great vessels is common. Or solidified exudation-matter may exist without adhesion occurring: this is particularly observed about the great vessels, where such matter may long (probably in perpetuity) remain the source of certain important physical signs above the heart's base. Locular or mesh-like adhesions, containing fluid, absorbable and renewable, sometimes form.

Thus terminates the disease, anatomically, in its acute stages; any ulterior changes, arising in the tissue or interior of the sac, clinically belong to, and will be described with, the chronic disease.

372. *Secondary lesions.*—Pericarditis, so frequently holding the position of a secondary lesion to other diseases, rarely entails anatomical change in disconnected tissues or organs. The various grave cerebro-spinal symptoms sometimes witnessed in the course of the disease are not assignable to any textural alteration in the nervous centres; they are purely dynamic.

Occasionally myocarditis, exudative and suppurative, occurs in the strata adjoining the pericardium; but here we have mere extension of a disease by contiguity of texture. The fibre of the heart sometimes rapidly falls into a state of local atrophy, giving place to intra-sarcolemmous fat,—but this can scarcely be regarded as a true secondary lesion. Endocarditis, frequently coexistent, is rather a joint effect of the cause producing the pericarditis, than a sequential result.

I doubt the bronchial inflammation, which occurs in a certain share of cases, being other than an additional effect of one and the same cause. But pneumonia does sometimes arise to all appearance, and pleurisy to a positive certainty, under the direct influence of pericarditis.

373. *Causes and circumstances of origin.*—Pericarditis is very rarely an *isolated* disease, and scarcely, except when traumatic, springs up in a person of sound constitution. The conditions,

under which it originates, may be grouped in the following manner:

1. Idiopathic.	
2. Traumatic	Wounds of pericardium from without, or through œsophagus;[*] foreign bodies from œsophagus;[†] blows and contusions of præcordial region.
3. Perforative	From liver-abscess through diaphragm,[‡] from cancer of œsophagus.
4. Attendant on or consecutive to—	
(*a*) Acute specific diseases .	Typhoid (Peyerian) fever, variola, scarlatina, pyohæmia.
(*b*) Diathetic diseases . .	Rheumatism, "Bright's disease," gout, scurvy, purpura, cancerous and tuberculous diatheses.
(*c*) Diseases of blood-vessels	Phlebitis, endosteal and other, after amputations and various operations.
(*d*) Peculiar states of the blood	Cyanosis [693].
(*e*) Sudden removal of long-established diseases	Psoriasis, chronic eczema, and lepra.
(*f*) Diseases of pericardium	Cancer, tubercle.
(*g*) Adjacent diseases . .	Pneumonia, pleurisy, pneumothorax,[§] inflamed mediastinum, abscess with probably diseased rib,[‖] costal periosteitis,[¶] abscess under abdominal surface of the diaphragm,[**] perihepatitis.
5. Terminal.	

374. A few words of comment on some of these various modes of origin seem called for.

(1) *Idiopathic.*—I have never seen idiopathic pericarditis as a solitary disease. But in those rare cases of acute bilateral pleurisy, occurring in previously healthy individuals, to which I have elsewhere referred,[††] pericarditis is a usual attendant, and has a claim to be considered idiopathic.[‡‡] It has, also, occurred to me to observe a case in which intense palpitation (160 to 180 systoles per minute), occurring without obvious cause, was followed by general dry pericarditis in the course of a week [723, *b*].

(2, 3, 4) *Traumatic, Perforative, Consecutive.*—Although indubitable examples of the various modes of origin, composing the above groups, occasionally occur, they are, all taken together, very rare; and, practically speaking, the complaint is scarcely met with in London but as a local development either of rheumatism or of "Bright's disease."

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[*] U. C. Museum, No. 3859,—Indian juggler's so-called knife "swallowed," and driven into the pericardial sac.

[†] Artificial teeth swallowed, lodging above the cardiac orifice of the stomach, and ulcerating through into pericardium.—Amer. Journal, Dental Sc., or Med. Times, May, 1858.

[‡] Graves, Clinical Medicine.

[§] Healey, U. C H., Females, vol. x., p. 91.

[‖] Newman, seen with Mr. Quain, U. C. H., Jan. 1854.

[¶] Llewellyn, U. C. H., Males, vol. xi., p. 174.

[**] Crawley, U. C. H., Females, vol. x., p. 18, March, 1854.

[††] Diseases of Lungs, 4th ed., p. 292.

[‡‡] C. Bäumler (Clin. Soc. Rep., vol. v.) relates some cases of "partial and general *idiopathic* pericarditis," adduced in support of an opinion that such inflammation of the pericardium is notably less rare than is commonly held.

Diathetic.—In rheumatic fever the joints commonly suffer before the pericardium; or the pericarditis may lead the way, the joints following; or both pericardium and joints may suffer simultaneously; or there may be no arthritic affection, pericarditis and acid sweats existing alone.*

The frequency, with which pericarditis forms a part of rheumatic fever, seems to vary in different localities, without its being possible to assign any special cause for excess in any particular district. The inflammation occurs among rich and poor, among the well-to-do and well-regulated members of the lower orders as among the most depraved; attacks young, middle-aged, and old; appears in cases of all degrees and forms of severity; accompanies every fresh attack of the fever in a given individual (I have known this up to the eighth), or omits an attack now and then; complicates rheumatism at all seasons of the year, and not more frequently during any one of the four than the others,—but does appear to occur more frequently, and to attain, as a rule, greater intensity, some years than others. The practical inference from all this is, that the physician should, under all possible varieties of circumstances, be on the alert for the detection of the disease.

John Taylor was the first to show that "Bright's Disease," in its advanced, as contradistinguished to its early, stages, acts with almost as much frequency, as rheumatic fever, in producing pericarditis.

Although pericarditis may by possibility occur as one of the secondary inflammations of typhoid (Peyerian) fever, it must be conceded the tendency thereto is extremely feeble, as Louis did not find the least trace of pericardial irritation in fifty-six fatal cases.†

Scurvy appears to act in some parts of Northern Europe as a very frequent cause of pericarditis. I have once met with the association in London, the scurvy having been caused by voluntary abstinence from vegetables of every kind for some years.

The cancerous diathesis very rarely acts as the efficient cause of pericarditis; even cancer of the mamma, so frequent a cause of adjoining pleurisy, seldom excites the pericardium to inflammation.

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† Perry, U. C. H., Females, vol. i., p. 82.
* Affection Typhoïde, t. i., p. 328, éd. i.

Nor does the tuberculous diathesis often evolve the disease; still I have seen it in phthisical persons (free from real tuberculous disease of the pericardium or heart itself, non-rheumatic, and possessed of healthy kidneys) under two forms. In the one the exudation-products are in no wise distinguishable from those of ordinary plastic pericarditis;[*] in the other those products partake of the characters assigned to true tuberculous pericarditis.[†]

Hambursin records four cases of pericarditis, in which the disease appeared in sequence to perihepatitis.[‡]

(5) *Terminal.*—Pericarditis, in this respect claiming a distant affinity to pneumonia, sometimes acts as the immediate cause of death in lingering diseases.[§] I have especially known it to do so in phthisis,—under these circumstances the serous inflammation has little of a diathetic character.

375. *Physical signs.* (*a*) *Dry stage.*—During this stage, the extent of visible impulse is greater than natural; the impulse, as felt, is too forcible, of beating rather than heaving character, and successive impulses are of unequal strength: the action is suggestive of excitement. The areas of percussion-dulness, both superficial and deep [70], are unchanged. Grazing friction-sound may occasionally be caught; and even sharp, though feeble, scraping sound, if there be a prominent tuft of dilated capillary vessels [131].[‖] The physician should, at this period, while it is yet unchanged by the disease, accurately ascertain the point of the apex-beat, to enable him to substantiate its subsequent displacements.

(*b*) *Plastic exudation-stage.*—Incipient bulging of the præcordial region may, it is averred by some observers, be now pretty constantly detected. In their apprehension "paralysis" of the muscular structure of the intercostal planes, induced by the adjacent inflammation, suffices, aided as it is by the protrusive action of the heart, to bulge the chest-wall. I have never succeeded in discovering this; nor does the theory seem at all sound. Admitting that the inflammation of a serous, in close contact with a muscular membrane, "paralyses" the latter, as in the alleged instance of pleuritis, it does not follow that pericarditis will have any such effect, seeing that the pericardium is not in contact with the side, and hence not with the muscular textures concerned.

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[*] Ryan, U. C. H., Females, vol. xiii., p. 43.

[†] Mercer, U. C. H., Females, vol. xiii., p. 7.

[‡] New Syd. Soc. Retrospect, 1871, p. 156.

[§] Healey, U. C. H., Females, vol. x., p. 91.

[‖] Parker, U. C. H., Males, vol. iv., p. 177. This is the only case I have ever met with where I succeeded in obtaining the post-mortem demonstration that mere excess of vascularity may generate well-defined friction-sound.

The inspection-signs are essentially the same as in the dry stage; but elevation of the apex may be effected by the contraction of lymph solely, if this stage be protracted [47, *b*]; the wings of the diaphragm may be somewhat raised too.

The hand sometimes detects pericardial friction-fremitus or jerking vibration.[*]

If the plastic exudation be very thick, it is conceivable that the area of superficial dulness shall be extended; but this is a sign too delicate and too uncertain to be trusted to.

The essential sign of this stage is pericardial or exocardial friction-sound, of which the properties have already been described [120 to 135] in all its clinical varieties.

The condition of the heart's sounds varies; they may be unchanged, or even louder than in health; or, on the contrary, masked somewhat by the loudness of the friction-sound, or even positively enfeebled, especially the first, in all probability by the interference of thick layers of lymph with the full play of the ventricles. I have known the second sound sharply reinforced at the pulmonary or second left cartilage.[†]

Valvular murmurs are of excessive frequency as dependencies on co-existent endocarditis,—especially the aortic constrictive and mitral regurgitant varieties. Simulative valvular murmur may, however, come directly of exudation on the pericardial surface. The aorta and pulmonary artery may be so pressed on by lymph, that murmur shall be engendered with the systole as the blood passes through the slightly constricted part [*vide* table of systolic right basic murmurs, 111, also 135 *c*.]. In regard of prognosis the possibility of such mechanism is obviously of importance,—a murmur, thus generated, must be of less serious import than one of endocarditic origin.

The respiration-sounds continue unchanged over the area occupied by the heart.

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[*] I have never observed fremitus produced by cardiac action on pleuritic lymph, the pericardium being positively healthy; Stokes, however, appears to have done so (op. cit., p. 29).

[†] Smith, U. C. H., Males, vol. xvi., p. 65, Feb. 19, 1859. But this man eventually left the hospital with a mitral regurgitant murmur.

(*c*) *Stage of liquid effusion.*—The perfection of the signs of this stage varies directly as the amount of fluid.

By *inspection* may be discovered arching of the præcordial region, widening and even bulging of its intercostal spaces, with elevation of the left edge of the sternum, sometimes traceably increasing from day to day, and in extreme cases bulging at the upper and left part of the epigastrium. Œdema of the præcordial integuments, especially if the effusion have existed for any time, is occasionally noticeable. Undulating impulse [47, *f*], and frequently displacement of the apex-beat upwards as far as the fourth interspace, and slightly outwards [47, *b*] complete the list of visible signs.

By *application of the hand*, added to auscultation, we find that in cases where the apex lies behind a thick stratum of fluid, the impulse lags slightly behind the systolic sound. The impulse feels weak, unequal, fluttering, or may be imperceptible; and if pericardial friction-fremitus had existed, it has now disappeared. The line limiting vocal fremitus at the right side of the heart is carried unnaturally to the right,—a valuable sign in some cases. The state of respiration-expansion over the heart varies,—if the quantity of serum be moderate, costal expansion is well marked, diaphragmatic movement being impeded by the fluid,—if very great, that expansion is impaired.

The interval by *measurement* between the left nipple and the middle line may be increased; but this is not always the case, even where bulging is well marked to the eye.

Percussion discloses that which is, all things considered, the most trustworthy sign of pericarditic effusion, namely, præcordial dulness of the peculiar triangular outline, the base below, the apex above, which has already been fully described [77, *c*]. The area of this dulness may be changed sidewards, most readily to the right, by moving the patient successively from one side to the other.

By *auscultation*, the irregularity of the impulse in regard of force, and, if this be affected, of rhythm, is better perceived than by other means. The friction-sound of the past stage may be either completely gone; or heard in some spots about the great vessels; or pretty generally retained in the præcordial region,—but this is very rare even with eight ounces of fluid, and seems scarcely possible with more than ten. On the other hand, *no conceivable amount of fluid will of necessity totally annul friction-sound.* I base this statement on a case, referred to a moment since, in which I and others distinctly heard friction-sound "at mid-sternum on the level of the third rib," and (yet death occurring only twenty-nine hours later) sixty measured ounces of fluid were found in the pericardial sac, which reached about a thumb's breadth *above* the clavicle.*

The possibility of systolic basic murmur being produced by pressure of fluid on the great vessels has already been referred to [135, *c*].

The heart's sounds feeble, distant, and as it were muffled, at the lower part of the cardiac region, become louder as the stethoscope is carried upwards, and at the top of the sternum the second sound is full and loud, and the first very decidedly more marked than directly over the ventricles. This is a sign which occasionally proves of great diagnostic significance.

Various displacements of adjacent structures occur. The anterior edges of the lungs are pushed aside by the accumulating fluid; they may by possibility be separated, on the level of the second cartilages, by a space five inches wide.*

Where copious effusion is suddenly poured into both the pericardium and the left pleural sac, it would appear, from a case observed by Stokes, the left lung may be pushed upwards so as actually to form a prominent elastic quasi-tumour above the clavicle.

The central tendon of the diaphragm undergoes depression, and may be rendered convex inferiorly,—producing slight epigastric prominence. And, further, as sometimes (but by no means always) happens, Duchenne's sign, in-sinking of the epigastric region with inspiration, may then be noticed. The liver *may* be pushed downwards and to the right, but an enormous amount of fluid will not necessarily displace it.

The respiration-sounds in the centre of the cardiac region are feeble and distant: in some very rare instances the voice resounds with an ægophonic twang at the edge of the effusion: this is especally likely to occur if the adjacent border of the lung be indurated.

(*d*) *Stage of absorption of fluid.*—True undulatory impulse now disappears,—the sufficing cause of *wavy* movement is no longer present. But a fluttering uneven impulse, by no means wholly dissimilar to the eye, may remain,—especially if the thoracic walls be thin. Nay, perfectly undistinguishable to the eye, occasionally in children: in a case seen at an advanced stage of the acute disease, and where the antecedent story was very imperfect, I have been completely deceived by the wave-like movement of a thin closely agglutinated heart. [47 *f.*] The point of the apex-beat almost invariably falls. The bulging of the cardiac region gives way.

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* Bartlett, U. C. H., Males, vol. iv., p. 292.

By the hand we ascertain that the impulse has recovered its breadth and fulness; friction-fremitus, too, may return.

The dulness of effusion gradually diminishes from above downwards, and draws in laterally also, but not till it has undergone a very distinct fall superiorly.

Redux friction-sound is caught by the ear, commencing commonly, but by no means always, about the roots of the vessels, and varying in extent with the rapidity of absorption. The churning or continuous rumbling variety is the rarest condition of friction-sound discovered.

The heart's sounds recover their fulness (if endocarditis have not prominently existed), and also their natural nearness to the surface. The præcordial respiration-sounds return slowly, and may never, especially if agglutination of the pleural surfaces occur in front of the pericardium, regain their natural intensity.

(*e*) *Stage of pericardial adhesion.* The occurrence of this stage is announced by disappearance of ordinary friction-sound, and *à fortiori* of friction-fremitus, if this have existed; the former may continue to be distinctly heard in some spots, where adhesion is as yet unestablished. Besides, the clicking variety of friction-sound may long continue audible at the base. The percussion dulness continues to decrease, or at all events does not increase in area. The action of the heart may be tremulous, unsteady, or jogging, and to the eye, as just mentioned, distinctly undulatory.

The moment adhesion is accomplished, the evolution of the acute disease has reached its final anatomical term.

376. *Symptoms.* I. *Local.*—The chief local symptom of pericarditis is pain, occupying the cardiac region only, mainly seated in the epigastrium, or extending to the left shoulder and elbow,—slight in amount or of agonizing severity, lancinating, tearing, burning, or consisting of a mere sense of soreness; and increased by movement, and deep inspiration. Pain, however, may be absent, and in the majority of cases is either absent or of slight severity. The varying amount of suffering in different cases is with difficulty accounted for; coexistent phrenic pleurisy seems to explain the acute agony of some patients, but pleurisy *may* be present and the pain moderate, or *vice versâ*. Rheumatism of the

diaphragm, phrenic or intercostal neuralgia or neuritis, are all conceivable, but non-proven, causes of excess of suffering.*

The præcordial interspaces are generally tender,—sometimes excessively so. Pressure in the epigastrium causes great distress in some cases; in others it is borne without a murmur. Bichat's overweening faith in the significance of the indications, derivable from such pressure, was one of those errors of observation into which men of the highest genius, when biassed by theory, will occasionally fall.

The action of the heart varies widely in intensity. There may be constant, more or less violent, palpitation, attended with many or few of the grave subjective miseries of that state; or there may be palpitation of irregular or regular rhythm, concerning the existence of which the patient is scarcely conscious; or the heart's beat may not, even to the observer, be appreciably abnormal.

377. II. *Systemic and Constitutional.*—(*a*) The decumbency is least commonly on the left side, most commonly on the back; the head is generally kept rather high. Orthopnœa, a most inconstant symptom, if present, is not any proof, as has been taught, that liquid effusion has taken place; it may be absent during the effusion-period, and first appear after absorption; again, it may occur paroxysmally and irregularly. Far from orthopnœa, or even raised position of the shoulders, being a necessary result of effusion, the patient, where this is most copious, may lie by choice flat on the back, with scarcely any pillow: this was the habitual posture, for days before death, in the case just referred to, where sixty fluid-ounces had accumulated in the sac. In such cases, the least elevation of the head produces a tendency to syncope,—and the patient instinctively dreads movement of any kind.† The facial expression is generally anxious; the features drawn: in fatal cases, *risus sardonicus* sometimes occurs towards the close. The sleep is fitful and disturbed; and jactitation of the arms—the trunk being kept quiet—not unfrequent in serious cases. Apprehension of death may be a prominent symptom.

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* The satisfactory demonstration by Luschka (Schmidt's Jahrbuch, 1853), that the phrenic is not simply a motor nerve, but contains sensory filaments distributed to the pericardium, as well as to the pleura, peritonæum, and abdominal wall, gives a definite character to the hypothesis of phrenic neuritis and neuralgia.

† Numerous examples, in addition to that referred to in the text, have fallen under my notice, showing that this is the habitual posture, when the quantity of fluid has passed a certain amount, the precise amount varying probably in different persons. What is the cause? It seems most likely that the posture is assumed simply because the circulation is so obstructed by pressure on the great vessels and heart, that instinctively the patient seeks the aid of gravitation to supply the brain and lungs with blood. These cases form a *syncopal species* of the disease.

(*b*) Rigors may announce the invasion of the disease, but they are not commonly severe nor repeated. Pericarditis acknowledges the law apparently regulating in the main the temperature in inflammations of the serous membranes,—that is, the thermometric rise is habitually null or very slight. Such pyrexia, as is objectively demonstrable, commonly depends on some associated disease; and pericarditis, supervening in the course of rheumatic fever, may not produce any, or at the most a very minute, elevation of temperature. Very great rise of heat, to 109° for instance,* and probably even higher, may certainly occur in acute rheumatism; but there is no reason to suppose the pericardial inflammation has any direct connection with the hyperpyrexia, whatever be the amount of evidence tending to affiliate the peculiarity to certain undetermined cerebral conditions. I have known the skin paroxysmally cold, and look upon this condition as of evil significance.

Such perspiration as occurs is not specially acid, unless rheumatism be present,—and it *may* be alkaline, even with this combination.† Sudamina sometimes form, and their contents may be alkaline, while the surrounding perspiration is acid,—or the reaction of both may be the same: none of these conditions are peculiar to the disease. Œdema sometimes occurs, particularly if the case be protracted, about the ankles; and may also appear in the integuments of the cardiac region. The integuments of the head, face, and neck, may become generally livid, with associated swelling of the medium-sized and large subcutaneous veins.

(*c*) The joints are not affected as a consequence of pericarditis,—they are frequently rheumatic, of course, as the majority of cases of pericarditis are of rheumatic origin. The limbs generally are the seat of such pains as appertain to an acute disease.

(*d*) There is nothing special in the state of the tongue. Deglutition, commonly natural, may be gravely obstructed in the manner to be presently [*k*, *l*] described. I have never met with a case in which pressure of the distended pericardium on the œsophagus even appeared to have acted as an efficient mechanical cause of dysphagia. Tenderness of the epigastrium, with nausea and vomiting of food, or of bile even (the stomach being perfectly healthy,‡) sometimes constitute very prominent symptoms—prominent enough, as in the case referred to, to throw all those of the cardiac class into the shade: epigastric pain, especially, is often decisively present, and is, sometimes, plainly connected with the phrenic nerves. The liver grows engorged; I have never seen jaundice. The bowels are confined.

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* Wilson Fox, *Lancet*, July 2, 1870. For full details, *vide* his work of earnest inquiry: "The Treatment of Hyperpyrexia," 1871.

† Numerous instances of this exist in my Hospital books.

‡ G. Perry, U. C. H., Females, vol. i., p. 82. 1846.

(*e*) Dry, irritable, abrupt, jerking, spasmodic cough, with variable dyspnœa, and coolness of the expired air towards the fatal close, are the chief pulmonary symptoms. That exaggerated respiration results from pericarditis *per se*, I more than doubt. The voice is sometimes much weakened, an effort required to produce feeble tones:[*] this symptom seems mainly connected with copiousness of effusion, though conceivably it may depend on reflex irritation of the vagus nerve.

(*f*) The pulse is commonly frequent out of proportion with respiration, unless there be some pulmonary complication. I have, however, known in rheumatic pericarditis, with chorea, a ratio of 136 : 64 or 2·12 : 1, the lungs being free from inflammation.[†] At first full and hard, and sometimes less frequent than natural,[‡] afterwards weak and feeble, the pulse grows irregular, both in force and rhythm, in about one-third of cases,—or such irregularity may exist from the first, and before fluid is present to account physically for the circumstance,—or irregular and unaccountable from frequency one day, it may be infrequent and perfectly regular on a subsequent one, the disease meanwhile advancing.[§] The frequency of the pulse is subject to more sudden variations, from the influence of emotional excitement and of effort, than in any other disease perhaps: thus gentle movement of the trunk may raise the rate from 80 or 90 to 130 or 140.

The sphygmogram in pericarditis, as may be gathered from these statements, presents nothing distinctive. The febrile state with which it is associated gives frequency to the curves, and well-defined dicrotism. During convalescence the pulse becomes infrequent, the curves in a series proportionally few and polycrotous.

The state of the blood of necessity varies in connection with the dominant diathesis; in so far as the pericarditis is directly concerned, the fluid is hyperinotic.

(*g*) The lymphatic system is unaffected.

(*h*) Having yet to observe a case of isolated idiopathic pericar-

[*] Bartlett, U. C. H., Males, loc. cit.

[†] Leason, U. C. H., Females, ætat. 9, vol. viii., p. 69.

[‡] Graves (quoted by Stokes, op. cit., p. 51) found the pulse "singularly slow" (infrequent) at the outset.

[§] Bartlett, U. C. H., Males, vol. iv., pp. 295, 6. Rhythm may be regular, while force is irregular.

ditis, I am unable to say what influence the disease *per se* exercises on the composition of the urine. As the complaint is daily seen, in association with diathetic and acute specific diseases, the urine is of highly marked pyrexial type, or possesses the characters of the disease to which the pericardial inflammation is superadded.

(*i*) I have not met with any special indications of sympathy on the part of the genital organs. Menstruation runs the same, but not a greater, chance of being arrested or deferred by an attack of pericarditis as of other acute inflammations: the function may be wholly unaffected.

(*k*, *l*) Slight cephalalgia is common;—the pain rarely sufficiently great to excite spontaneous complaint. As a rule encephalic symptoms of any serious import are totally absent from first to last. But, on the other hand, delirium, apoplectiform stupor with imperfect hemiplegia and a quasi-maniacal state* or actual mania of violent character, sometimes give special character to the case. And grave nervo-muscular disturbances, pointing to spinal origin, are occasionally witnessed: among these may be enumerated laryngismus; external cramps; slight clonic spasms, scarcely amounting to fits of convulsion; epileptiform, hysteriform, trismal and tetanic seizures, and chorea in all degrees of severity.

These cerebro-spinal symptoms are sometimes in all likelihood produced rather by the altered state of blood coëxistent with the pericarditis than by the local conditions of this inflammation in itself: in the pericarditis of "Bright's disease" uræmia is doubtless their immediate cause. In the rheumatic variety these symptoms may, however, be of reflex mechanism, excited by irritation of the phrenic nerves,—a view, though deficient in proof, certainly more tenable, than that ascribing them to cerebro-spinal meningitis: for the positive absence of that inflammation has more than once been demonstrated *post mortem;* and when it actually exists, no one of the symptoms just enumerated may occur.† Since I originally noticed dysphagia as a symptom of pericarditis in the first edition of this work, I have seen some five or six additional very striking examples: ‡ in one of the patients, an adult, wildness of manner, tremors, paroxysmal orthopnœa, with faintness and subsultus tendinum coexisted; in the other, a child, violent bilateral choreal movements and imperfect laryngismus. Both cases confirm the opinion expressed in a former place [372], that the symptom is of purely dynamic mechanism.

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* Campion, U. C. H., Females, loc. cit.

† Gash, U. C. H., Males, vol. x., pp. 178—182.

‡ *e.g.* Campion, U. C. H.. Females, vol. vii., p. 260; and Miss B——, seen with Dr. Neil Arnott. My statement appeared in the edition of 1851 (p. 443), and I believed it wholly novel. I have since learned, however, the symptoms had been distinctly described by Testa in 1811. The fact was certainly not acknowledged by the English or French medical schools in later days.

(*m*) Alterations of the functions of special sense have not fallen under my notice, unless in a few cases where they (blindness and deafness) were plainly referrible to the dominant diathetic state present,—that of "Bright's disease."

378. *Course and Mode of Progress.*—Pericarditis may run a uniform course, with its several periods of increase, status, and decline. Or the disease may at any moment undergo temporary suspension, not only in respect of its subjective symptoms, but of many of its objective conditions. True relapse of pericarditis is certainly uncommon; whereas, as far as I have seen, recurrence is more frequent than in the case of any other inflammation of grave character.

In the great majority of cases the symptoms of pericarditis are sufficiently marked to draw the attention of both patient and observer to the seat of disease; in some few, however, all subjective indications are wanting,—the affection, completely latent, is only to be detected by objective evidence. Such latency of course is more common in the diathesis of "Bright's disease," than under other circumstances; but I have a considerable number of cases recorded in the hospital books, where even in rheumatic fever all the physical signs of exudation and of moderate serous effusion existed without the slightest pain or functional disturbance of the heart. Latency of course may occur under all anatomical conditions of the disease,—and seems plainly connected with the special nervous susceptibility of the individual, rather than with the amount of local mischief.

379. *Terminations.*—(1) When acute pericarditis terminates clinically by *recovery*, what are the anatomical changes effected? John Taylor thought strata of lymph might be *completely* absorbed; his evidence, however, simply amounts to this,—that, as friction-sound sometimes disappears without signs of adhesion ensuing, lymph, which must have been the cause of that friction, has been absorbed. Now, as it is certain friction-sound may be produced by mere vascularity of the membrane [375, *a*], the whole argument falls to the ground. Still, there seems no objection to admitting that such absorption on a limited scale may sometimes occur. But, be this as it will, the *common* terminations are by absorption

of fluid, with adhesion of the serous surfaces,—or without such adhesion, where lymph has been exuded by one of the two surfaces solely.

(2) Or instead of recovery, in the complete sense, occurring, the acute passes into the chronic disease, either with adhesion and agglutination, and more or less constant tendency to irritative congestion,—or with persistent effusion.

(3) Or death occurs. Commonly syncopal, death may be *gradually* effected either by the mechanical interference of the fluid with the heart's action, or less readily by that of effused lymph. It seems wonderful what an amount of pressure by accumulated fluid can be borne with impunity by the heart. Fifty to sixty ounces do not necessarily stop its contractions; but this obviously depends on the slowness of its accumulation and extensibility of the pericardium: a few ounces of blood suddenly poured out from an aneurism into the serous cavity will, we know, mechanically destroy life in an instant,—still, here, the nervous shock of the arterial rupture plays some part in the catastrophe. Contraction of effused lymph may so twist and distort the heart, that mechanical suspension of projection of the blood through the orifices becomes well conceivable.* Besides, there can be no doubt intrinsic changes in the heart's own muscular condition—dynamic failure and alteration of texture by acute interruption of nutrition—occasionally help to arrest its movements. Or death may *suddenly* take place from raising the head or otherwise moving abruptly,—an acute syncope caused by cardiac paralysis. Lastly, the fatal event may be mainly traceable to some one or more of those forms of cerebro-spinal disorder a moment since described.

380. *Duration* (*a*).—In rheumatic pericarditis death rarely occurs before the tenth or twelfth day; and may be deferred for upwards of three weeks. On the other hand, in less than thirty-six hours life may be extinct. Andral records an instance fatal in twenty-seven hours from the first supervention of pain. The great orator of the first French Revolution, Mirabeau, was cut off by pericarditis so rapidly, that the reports of his having been poisoned readily gained credence.

(*b*) In instances of recovery, disappearance of subjective, and of most of the objective, symptoms takes place in from twelve to twenty days; but physical signs may hold on for weeks, nay, for months, especially slight percussion-dulness, and clicking friction-sound [120, *c*], above the heart's base, while ordinary friction may occasionally continue audible towards the apex and over the general surface [130].

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* Campion, U. C. H., Females, vol. vi., pp. 40, 41; also Mercer, U. C. H., Females, vol. xiii., p. 4.

381. *Diagnosis.*—The diagnosis of pericarditis can only be made with security by means of the physical signs. For, first, so absolutely *latent* from the beginning to the end may the disease prove, that I have known patients with several ounces of fluid and exudation-matter in the pericardium, grow irritated, when inquiries were made about symptoms connected with the heart; * secondly, there may be a total want of harmony between the violence of the symptoms of which the patient is actually conscious and the amount of disease; or, thirdly, the symptoms of other affections may be simulated.

Before the occurrence of friction-sound there is no certainty in the diagnosis. But if there coexist with sudden excitement of the heart and absence of endocardial murmur præcordial distress and tenderness under pressure, while the pulse is frequent (I have not been able to confirm Graves's observation as to its initiatory infrequency), and the respiration non-accelerated, the probabilities pronounce for pericarditis. This will especially be true, if any one of the diathetic or acute specific diseases, with which pericarditis is known to coexist, has preceded.

The essential signs of pericarditis are friction-murmur, special præcordial dulness, and twisting upwards of the heart's apex. Let us inquire into the conditions and surety of their diagnostic significance.

(*a*) Friction murmur, when thoroughly developed with the characters assigned it elsewhere [120, 130], is next to pathognomonic: the possible fallacy from pleural friction of cardiac rhythm must not, however, be forgotten. Mediastinal pseudo-rhonchus (which may coexist, too, be it remembered, with true pericardial friction), is another possible source of fallacy.† By a little care the squashy rhonchoid sound, sometimes produced by pressure of the stethoscope over the heart (as on other parts of the surface), when the integuments are œdematous, will be distinguished from pericardial rubbing-sound; its rhythm is of course respiratory.

I have nothing to add here to the several rules already laid down for the distinction of endocardial from pericardial mur-

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* This kind of pericardial anæsthesia (if I may use the expression) is particularly prone to occur in patients affected with "Bright's disease."

† Diseases of Lungs, 4th Ed., p. 123.

murs [133]: not very unfrequently troublesome, occasionally impossible, the distinction is, on the other hand, in the mass of cases easily effected.

It must not be forgotten that a certain murmur, really intra-pericardial, may be mistaken for other things. This is, perhaps, especially true of the clicking variety of pericardial friction [120, *c*]: this differs, however, from *valvular* clicks in its non-synchronism with either heart-sound, and in its non-transmission along the aorta and pulmonary artery.

Again, exudation may be present, and yet no friction-sound evolved,—either because the posterior aspect of the heart only is affected,—or because one lamina of the membrane only is coated with lymph,—or because recent agglutination has occurred,—or because old agglutination prevents attrition,—or because serum or pus has been copiously poured out. And to all these causes of the non-occurrence of attrition must be added the mechanical conditions, as they may be called, of the lymph itself; if this be particularly smooth, and slimy to the feel, no friction-murmur will arise.*

(*b*) Next, of the significance of the percussion-results. If the peculiar triangular-shaped dulness be plainly developed under the eye of the physician, and have succeeded to friction-murmur, there is no possible source of fallacy. But if it have not been preceded by friction, the dulness may depend on hydropericardium simply; and if the triangular form have not been produced under observation, there are certain sources of fallacy of a serious character already glanced at [77, *c*]. For instance, a weak fatty heart, with quasi-undulatory impulse and feeble sounds, intermittent pulse, and febrile action, may coexist, where the form of the heart's dulness is rendered triangular by the chance presence of old exudation-matter about the great vessels, a tumour, a small solid quiescent aneurismal sac,—or even of an excess of natural mediastinal fat. How is the distinction of the cases to be established? Probably, in the whole range of thoracic diagnosis, there does not exist a more difficult problem. This truth is exemplified by the following case. An adult, labouring obviously under acute disease, unable to give any trustworthy account of himself, had all the physical heart-signs mentioned, and in addition orthopnœa and jactitation; while, on his admission to the hospital, no ordinary signs of pneumonia or other thoracic inflammation existed to account for the acute aspect of the disease. I was strongly disposed to regard this as a case of latent pericarditis with effusion, accompanying valvular disease, of which latter affection the signs were obvious; but the impossibility of discovering a shadow of friction-sound, though the patient's posture was varied, the non-elevation of the heart's apex, and the fact that above the third rib the dulness was not so absolute as below it, led me to reject the idea. On *post-mortem* examination (the signs of pneumonia had meanwhile made their appearance,—on admission, indeed, the pulse-respiration ratio was 2·7 : 1, and in twenty-four hours had become 2 : 1), the pericardial sac was found free from fluid; but above the base of the heart lay a lump of fat, the simple source of all the difficulty.* And it is quite conceivable this difficulty might be increased;—for a patch of old exudation-matter near the apex might give friction-sound, while all the other conditions remained essentially the same as in the case just referred to.†

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* Mercer, U. C. H., Females, vol. xiii., p. 4, January, 1857.

Mediastinal tumour of some size, if it encroach on the cardiac region, may simulate pericardial effusion. But the history of the case; the presence of centripetal pressure-signs, which are never caused by fluid in the pericardium: and the outline of the dulness, which can scarcely by an unlucky chance imitate precisely that of pericardial dulness, (a tumour grows in spite of gravity, fluid obeys this,) will distinguish the former from the latter. Still the diagnosis must occasionally be profoundly difficult: for Skoda refers to a case, where a trochar was driven into an encephaloid mediastinal mass, under the impression that the instrument was to give issue to pericardial fluid. *Per contra* it may happen that intra-pericardial fluid imprisoned by old adhesions about the great vessels at the base of the heart, and limited to that locality, shall to a certain extent simulate a solid mass in the anterior mediastinum.‡

(*c*) If the elevation of the heart's apex occur during observation, I believe it to be an infallible sign of pericarditis; simple hydropericardium has never within my experience sufficed to displace that point to any notable amount. But I think it right to warn the student, that, where the change is not actually effected under observation, he must be on his guard against a certain number of sources of fallacy. The chief of these are, as follows:—the heart's apex, either as a result of original conformation, or through the long-continued influence of stays, may lie unusually high; it may be raised by the contractile influence of the exudation-matter of a long-past attack of pericarditis; it may be pushed upwards and outwards by an enlarged liver; or upwards and inwards by an enlarged spleen; or it may be drawn aside and upwards by the contraction entailed by old pleurisy; or it may be drawn upwards a space and a half by a contracting excavation in the upper part of the left lung; and lastly, a slight but real elevation of the heart's apex may take place through retraction of the great vessels and shrinking of the heart's cavities consequent on great hæmorrhage. As blowing murmur will exist in the latter case, an erroneous assignment of the position of the apex to the influence of pericarditis is the more likely to occur.

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* Beckett, U. C. H., Males, vol. v., p. 229.
† J. Morris, U. C. H., Males, vol. vii., pp. 154—162.
‡ Richmond, U. C. H., Females, vol. xv., p. 5, December, 1858.

(*d*) There are, besides, particular combinations of *symptoms*, occurring in pericarditis, which simulate with considerable closeness those of very different affections. Here is an instance. A female had fever, dry red tongue, extreme epigastric, and no præcordial, tenderness, bilious vomiting and diarrhœa, and perfectly regular pulse; she presented not a single symptom, subjective or objective, connected with the heart, and was free both from "Bright's disease" and from acute affection of the joints. How is this state of things, which proved to be really dependent on pericarditis with effusion, to be distinguished from acute gastritis? The physical signs say nothing; friction-sound has already disappeared when the patient is first seen, and a huge stratum of subcutaneous fat with a massive mamma may deprive us of the evidence derivable from percussion.*

(*e*) Again, important though the existence of one of the diatheses or cachexiæ, to which pericarditis is commonly linked, be as an element of its diagnosis, the absence of one or of the other must not negative the positive evidence of physical signs. Some years since it occurred to me to observe a case in which every possible sign of pericardial effusion clearly existed; but I declined to place the diagnosis, "pericarditis," on the hospital bed-ticket, because the patient's constitution was sound, no injury had been inflicted on the chest-wall, and she was free from rheumatism. This was the instance referred to in the table of causes [373, *g*], where an abscess under the central tendon of the diaphragm had been the exciting cause of the pericardial inflammation really present, as proved *post-mortem.*[*]

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* Facts, all of them, illustrated by the case of Perry, U. C. H., Females, vol. i., p. 82.

(*f*) In the diagnosis of a difficult case, the functional and general symptoms should not be forgotten,—but clinical experience compels us to admit that they are sometimes utterly fallacious aids. Hope held the singular creed that "the variability of the symptoms is calculated rather to enlighten than perplex the practitioner,"—his belief being that the symptoms of lymph-deposit were slight, of effusion serious. The value of this dogma appears from a single case, the fourth in Andral's collection, where death occurred from plastic percarditis in twenty-seven hours, without a drop of fluid having formed in the sac; while, *per contra*, the disease may be latent with extensive hydro-pericarditis.

382. *Mortality and Prognosis.*—There are no existing documents from which the absolute mortality, or the ratio of deaths to seizures, may be safely calculated on a large scale.

The *immediate* prognosis is in the majority of cases not unfavourable. Louis long since estimated the deaths at one in six of those attacked. But the result is greatly under the influence of the existing diathesis. I do not remember to have seen rheumatic pericarditis fatal, in a previously healthy person, except in some seven or eight instances; whereas a large proportion of cases of pericarditis, arising in "Bright's disease," end fatally. Taylor found in seventeen cases of recovery the mean age = 19·7 years; in twenty-one cases of death = 35·5 years: but renal disease would be more likely to exist in the series of more advanced age than in the other,—age alone cannot, therefore, be charged with the difference in mortality. The whole class of reflex phenomena are of excessively bad augury: when highly marked choreal symptoms have occurred in childhood, I have never known recovery ensue. Dysphagia at any age is of very fatal significance.

In rheumatic pericarditis sudden and great rise of temperature is of the very gravest augury: whether the hyperpyrexia, it signifies, be connected or not with manifest intracranial complication, the general truth remains the same, that an abrupt rise to 107°, *à fortiori* to 109°—110°, is almost surely followed by speedy dissolution. The temperature kills.

In regard to *remote* prognosis: if mere adhesions or even agglutination exist, the health does not necessarily suffer; if hypertrophy, simple or dilated, or atrophy supervene, symptoms of course arise. Beau found in forty-eight cases of agglutinated pericardium forty examples of dilated hypertrophy, but the relative dates of the two conditions cannot always be established from his facts. I have seen no reason to accept the doctrine that old pericarditis in itself kills: coexistent endocarditis with its valvular sequelæ is the real destroyer in chronic fatal cases [398].

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[*] Crawley, U. C. H., Females, vol. x., p. 18, March, 1854.

383. *Treatment.*—In its character of an acute inflammation, affecting sometimes directly, always indirectly, the dynamic and the statical conditions of the heart itself, simple pericarditis would theoretically call aloud for antiphlogistic treatment of a most active kind. But as matter of actual experience we find that, while isolated idiopathic pericarditis scarcely ever exists, the form of diathetic state, on which the disease supervenes, forces us very materially to modify any therapeutic system, founded on the mere idea of phlogosis.

384. In order to avoid hyperdivision, we may consider pericarditis *quoad* treatment, as referrible to two varieties only: a variety belonging to rheumatic fever, and a variety in which the inflammation appears as a local expression of some established cachexia. The latter variety may be considered to be typified by the pericarditis of "Bright's disease."

Now the absolute necessity of distinguishing these two varieties flows from the fact, that rheumatic pericarditis in the great majority of cases terminates favourably, no matter what the precise mode of scientific treatment adopted; whereas actively acute pericarditis of renal origin almost invariably proves fatal.

385. (1) *Rheumatic pericarditis.*—(*a*) *Sthenic.* Whether the pericarditis appear after, along with, or before the articular affections, possibly the intensity of the pericardial inflammation might be lessened by artificially irritating the joints. To grant this obviously does not require one to have any faith in the exploded doctrine of metastasis; but the *practice* has disappeared, perhaps unmeritedly, since the *theory* has been banished from the schools.

386. *Blood-letting* claims consideration foremost among professed remedial agents in rheumatic pericarditis. Let me commence this inquiry by observing that the following truths show the wisdom of caution in the amount of blood abstracted. Bleeding from the arm cannot be pushed to any notable extent without the risk of inducing syncope,—an occurrence of serious danger when already the heart's vigour is dynamically and statically impaired. Venesection, be it ever so free, not only does not *of necessity* arrest, or very sensibly modify, the course of

the existent pericarditis,—but may actually fail to prevent the development of other inflammations (for instance, pneumonia,) secondary to itself. Very copious depletion, particularly in some constitutions, excites the heart greatly, and in several respects makes matters worse than they were. And, further, the most severe rheumatic pericarditis may go on equably or interruptedly to recovery, though very moderate cupping or leeching over the præcordial region have been substituted for profuse phlebotomy. Yet, on the other hand, I must candidly own that, to my apprehension, the query, whether bleeding at the very outset may ever succeed in arresting all phlogistic action at once has never yet been scientifically answered in the affirmative or the negative. Instances in which the inflammation is positively caught in the nascent state are rare; and if treatment appeared to have attained its object, the accuracy of the diagnosis at so early a period of the disease might reasonably be questioned. Meanwhile it is certain, lavish depletion in some cases lessens pyrexia and calms local pain and distress, not only without preventing, but without a jot abating the activity of, the exudation- and effusion-processes. Meanwhile, too, that the directly curative importance of copious blood-letting has been exaggerated, while its special dangers were ignored, no reasonable doubt can be entertained. In the fulness of his enthusiasm, Hope wrote, "*the loss of a few hours at first may be irretrievable, and hence hesitation and indecision may seal the fate of the patient. Venesection to the verge of syncope, followed by twenty-five to forty leeches,*" he holds to be essential in severe recent cases; and "unless the pain be completely subdued by these measures, the leeching, and in some cases the general bleeding also, may be *repeated two, three, or more times.*" What then is to be said of a case, which I have elsewhere published, where violent acute rheumatism coexisted with pericarditis, endocarditis, double pneumonia and pleuritic effusion, and probably aortitis, and yet, twenty-two ounces only of blood having been taken by cupping from the cardiac region, convalescence was established by the twenty-first day?* I never draw blood largely from the arm in rheumatic pericarditis; and the reasons why I abstain from the practice are not that I fear people profusely bled shall "forthwith go raving mad" (as, according to the statement of Latham, they nevertheless have, though in rare instances, actually done); nor that I fear endocarditis shall forthwith be generated,—an effect which has by some authors been positively assigned to the practice, but which effect the said practice much more certainly has never been proved to entail. My reasons are, first, that none of the advocates of such venesection have ever shown that the mortality, duration, or suffering, of the disease, are less in a series of cases thus treated than in a series of cases treated by gentler means—by aconite, colchicum, alkalies, and aperients, for instance. And secondly, that while, by prodigal bleeding, a loss of vital fluid is inflicted, which it may take the nutritive system months, or even years in individuals of certain constitutions, to repair, all that loss may be saved without demonstrated risk of any kind.

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* Clinical Lectures, case of Craddock, Lancet, February, 1849. I have repeatedly seen this man since 1849 with relatively slight attacks of rheumatism. In the intervals of these seizures his health and muscular vigour are perfect,—and such as I do not imagine he could have regained had he been lavishly bled.

On the other hand, that moderate venesection, as matter of experience, shortens the duration of pericarditis, and does so more effectually the earlier it is performed, has been clearly shown by Taylor in his logical papers on the treatment of the disease. The quantity of blood to be drawn must be regulated by the severity of the symptoms: from an adult of medium strength some eight to twelve ounces may be taken from a vein in the arm, the head being kept *low*, especially if there be much fluid in the sac. This depletion may be followed, if well supported, by the abstraction of some six or eight ounces more by cupping or leeching over the heart.

I have known moderate blood-letting improve, within a few hours, the tone of the first sound, probably by exciting absorption of fluid in the sac, and facilitating cardiac action.

In cases of medium and of slight severity, venesection may, with advantage, be replaced by the application of a few leeches to the præcordial region, or by the removal of blood to the extent of four or six ounces by cupping. And, as far as my own experience of late years is concerned, it would appear that cases of medium and slight severity must, to my apprehension, greatly outnumber those of graver character; for a considerable time has passed since, as far as I remember, I have employed any more active depletory measure than the application of some eight to twelve leeches.

Such are the inferences to which I have been led by, as I believe, unprejudiced experience concerning the abstraction of blood in acute pericarditis. I make no allusion to the controversial papers on the subject that a while since engaged so large a share of professional attention. Not that I am wanting in respect—in some instances even admiration—for the ingenuity and subtleness in argument displayed by some of the disputants; but that, I firmly hold, practical questions, are not to be decided by scholastic exercitations. Just as in theological casuistry a successful disputant proves himself more "cunning in fence" than his beaten adversary, but neither demonstrates the truth of his own, nor proves the unsoundness of the rival, doctrine,—so, in clinical *à priori* disputations, truth may really lie with him who is utterly worsted in argument. In all that relates to the practice of our art, experience alone can guide us, with any approach to safety, through our difficulties; the rest is *vox et prœterea nihil.*

387. Although much has been more or less eloquently written on the powers of *mercury* in rheumatic pericarditis, especially on the faculty it possesses of causing the absorption of, and repairing the damages effected by, exudation-matter, it must, I think, be conceded, that any precise evidence before the profession fails to demonstrate the alleged prowess of the mineral.[*] Pytalism is with difficulty induced; and when induced does not, of necessity, arrest the exudation- or effusion-processes. Taylor notes that in three of his cases (no very small proportion of the whole number) increase of signs and symptoms manifestly followed salivation; and, it is inferrible, he observed many others wherein such increase, though less in amount, was not less real as matter of fact. Taylor's data indeed, taken as a whole, distinctly argue against the efficacy of mercury. He refers to the frequent occurrence of sundry acute inflammations during the progress of salivation, induced for the intended cure of others. For my own part, I have known pericarditis supervene in a woman while under treatment for ptyalism, so severe, that for some hours after her admission into hospital for the cure of that ptyalism, life was in danger from semi-asphyxia, and tracheotomy on the point of being performed.[†] Mercury is supposed to act in inflammations by diminishing the quantity of fibrine, yet Andral found hyperinosis in four cases of mercurial stomatitis:[‡] it surely will not be con-

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[*] "If a person is seized with very acute pericarditis *how unavailing will be our best-directed efforts, unless they be succeeded by a speedy mercurialization of the system.*" But Graves, the writer of these words, gives no proof justifying trust so implicit. The observer of to-day, musing over the quasi-passionate faiths of Hope and Graves in blood-letting and mercury, will probably smile or sigh, as temperament may have made him a disciple of the laughing or of the weeping Philosopher of old; but he may in either case accept a warning from the errors of two great physicians to avoid the self-deception, into which therapeutical enthusiasts are so readily betrayed.

[†] Spratt, U. C. H. Females, vol. iv., p. 471.

[‡] Hématologie, p. 90.

tended that herein we have an illustration of the dogma *similia similibus.*

I must admit that year by year my faith in mercury has waxed less strong,—and that a long period has elapsed since I have made even a deliberate attempt to salivate a pericarditic patient.

388. On the principle of regarding the disease through its diathesis, *alkalies* are advisable, where the pericarditis is rheumatic. Besides, their influence on the inflammatory exudation-process *per se* seems more active, as taught sixty years ago by Mascagni, than that of other known agents. It was formerly thought the use of *colchicum* should never be omitted in a case of this kind, but of late years belief in the real efficacy of this agent in rheumatism has greatly lessened. In overdoses it may induce sudden and dangerous failure of the heart's activity. *Opium* becomes a necessary remedy in full proportion, should agitation and disquietude be at all marked; morphia may be used endermically, or hypodermically in cases of acute agony. *Veratrum viride*, *digitalis*, *hydrocyanic acid*, *et alia similia*, are dangerous agents, from the chance of their increasing tendency to syncope,—whether spasmodically or paralytically. *Purgatives*, *diuretics*, and *diaphoretics* are advisable as aids in the treatment.

389. *Sinapisms*, frequently repeated, are of great service in relieving pain and distress. *Ioduretted frictions*, coupled with *mercury* in very small proportion, seem to promote absorption of exudation-matter, especially when the more acute state has passed by.

The *hot-air or vapour bath* is a valuable adjuvant,—both have the advantage over the warm bath, that they may be taken with the head moderately low.

On the other hand, when sudden hyperpyrexia occurs in rheumatic pericarditis, the existence of effusion, however strongly suggestive, as an ordinary rule, of extreme caution in moving the patient, does not outweigh the urgent motive on other grounds for the use of the *cold* bath.* The risk of raising the head and of keeping it raised for a while, is to a certain extent limited and problematical; while the tendency to death becomes so strong, if the hyperpyrexia be not cooled down, as to render a fatal issue well nigh inevitable.

390. The *regimen* must be essentially antiphlogistic; as a matter of no small importance, the patient should be strictly cautioned, if there be much fluid in the sac, against abrupt

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* Wilson Fox, Lancet, July 2, 1872.

movements and elevation of the head. In cases where failure of the heart's force occurs, whether early or late in the attack, animal food, diffusible stimulants and brandy are called for. Even though this failure come of actual inflammation of the heart's fibre, the pulse must be sustained by alcoholic stimulus.

Blistering the præcordial region seems to act beneficially by relieving pain, lessening deep-seated irritation, and promoting absorption. When it is of importance to watch with precision the progress of events from day to day, vesication had better be effected on the confines of, than exactly on, the cardiac region.

I feel satisfied that if pericarditis *be caught at its very outset*, if the pyrexia be well marked, and the general conditions sthenic, *tincture of aconite* in small and frequently repeated doses (two to four minims, P. B.) may be given (*fas est et ab hoste doceri*) with a well nigh certainty of lessening the frequency and force of the pulse, lowering the temperature, and possibly controlling the inflammation. Cautiously used, it can do no harm; but in over-doses it will accelerate, instead of slackening, the rate of the heart's motion, and dangerously lower its power.

The almost too wonderful results announced concerning the efficiency of *propalymine* in acute rheumatism (Awenarius), leave its influence on endo-pericarditis in an unsettled state.*

No special treatment is required for the various inflammations sometimes occurring secondarily. But the whole class of reflex symptoms call for the suspension of directly lowering measures and of mercury, if these have been employed; purgatives, anti-spasmodic and sedative remedies must at once be had recourse to; and an attempt made to excite irritation, as rapidly as possible, in the joints by sinapisms. If dysphagia arise, the neck should be blistered.

If the statements concerning the treatment of the disease now set forth be in accordance with sound experience, it would follow that our demonstrable means of seriously modifying the progress of acute pericarditis are exceedingly limited. And I acknowledge this to be my conviction,—nor do I stand alone in holding it. In a recent essay (Clin. Notes on Pericarditis), T. W. Gairdner advocates a system of treatment which seems closely allied to "pure expectation;" and he "has not had under his care a case of rheumatic pericarditis fatal during the acute period of the attack." *His* faith cannot be strong in active measures.

Surgical interference in the acute conditions of the disease is, in the vast majority of cases, wholly inadmissible. But when fluid rapidly accumulates, producing quasi-syncopal conditions which jeopardise life at each moment, *paracentesis pericardii* may be resorted to. The operation has occasionally saved life* [401].

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* Brit. Med. Journ., Jan. 1873.

390*. (*b*) *Asthenic.*—In asthenic rheumatism, no matter from what sources the asthenia springs, pericarditis should not be treated antiphlogistically. Carbonate of potash and of ammonia internally, counter irritation, good nourishment, and moderate doses of wine or brandy, form the staple agents of treatment. If the notions of the late R. B. Todd were accepted, the administration of brandy should be conducted with a lavish hand, not only in the present, but even in the markedly sthenic, variety of the disease. But to my mind his argument fails—even as an argument—to carry conviction with it; and the fact that a young female, labouring under violent pericarditis, may absorb a bottle of brandy *per diem,* and recover, seems to me simply to show how difficult it may prove to overcome the tenacity of life with which Nature in rare instances endows us.

391. It may not be unwise to suggest for the thoughtful consideration of those who now occupy the benches of the schools, and many of whom are disposed, with the enthusiasm incident to youth, to become the future practical exponents either of the extreme antiphlogistic, or of the extreme stimulant doctrine, that exaggerated courses of procedure in dealing with complicated and but partially intelligible derangements of vital actions, are, on logical grounds, gravely wrong. Neither he who pours away the blood of his patient and dreads stimuli as deathly, nor he who looks upon the sacrifice of an ounce of blood as seriously detrimental, and reverences brandy as alone restorative, *scarcely knows even in a fragmentary and imperfect manner the immediate and intimate effects of the methods he employs.* On the ground then that a given cause will produce a certain given effect, and none other, these extreme measures are indefensible,—especially as the unknown portion of the effects, where the cause is so powerful, may, for aught that is proved to the contrary, be more or less disastrous. Suppose, however, changing the ground, that one faction of these ultra-therapeutists, professing total indifference to the nature of all immediate and intimate vital changes induced by their method, point exultingly to their results, and demand to be judged by these; the other forthwith prove the vanity of the exultation by

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* *Exempli gratiâ,* Albutt, Med. Times, Nov. 1866.

proclaiming the success of their own. The two groups of results, in their quality of evidence supporting one or other method of treatment, obviously destroy each other. And so the fancy intrudes itself, that perchance in both series Nature has been the real victor over both disease and treatment.

392. (2) *Pericarditis of "Bright's Disease."*—Bleeding to any extent in the pericarditis of "Bright's disease," occurring as it mainly does in the advanced periods of that affection, when the blood is deeply tainted and the nervous vigour impaired, is even theoretically contra-indicated, and as matter of unfortunate experience, has been shown to be deeply injurious. Extreme activity of the congestive and exudative processes may, however, be somewhat controlled by the abstraction of two or three ounces of blood from the cardiac region by leeching or cupping. Counter-irritation by blisters, applied in the neighbourhood of the heart, is also advisable.

General opinion seems to me to have wisely decided that the administration of mercury is, on the whole, injurious in all diseases attended with albuminuria,—and especially in the group of diathetic affections included under the head of "Bright's disease." Iodide of potassium and bicarbonate of potash, in full watery solution, are probably the medicines best calculated to act diuretically in removing the uræmic poison from the blood, which may fairly be regarded as the exciting cause of the pericarditis. The hot air, or the vapour bath, and hydragogue purgatives will favour elimination by other channels. Dry cupping over the kidneys, by lessening congestion, promotes the secretive activity of those organs.

B.—CHRONIC PERICARDITIS.

393. Under the head, chronic pericarditis, may be included four states clinically very different: namely (*a*), that in which adhesions or agglutinations of the pericardium having formed, a tendency to active congestion in the pericardium itself, and in the substance of those adhesions, is more or less constantly present;* (*b*), that in which cardiac hypertrophy is conjoined with such adhesions; (*c*), that in which cardiac atrophy co-exists with such adhesions; (*d*), and that in which liquid effusion remains in the sac without apparent inclination to increase or to disappear.

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* Vessels in old adhesions will sometimes bleed freely on being torn across,—*e. g.*, Mann, U. C. H., Females, vol. ii.

(*a*) SIMPLE PERICARDIAL ADHESION.

394. In the first form, that of adhesion, there may be, in the absence of irritation, a complete nullity of symptoms,—neither in cardiac action subjectively considered, nor in the state of feeling about the heart generally, is there anything to arrest attention. If there be congestive tendency, palpitation is easily excited, the breath short, and uneasiness of various kinds readily induced.

395. *Physical Signs.*—We have seen what the physical signs of adhesions are at the time of their formation [375, *e*]; enlightened, as the observer then is, by the previous course of events, they are easily established. But in a case, seen for the first time, after adhesions have been some while formed, their positive diagnosis is among the most difficult clinical problems existing. Nay, more, I believe the achievement an impossible one in a goodly proportion of cases: I have known adhesions found after death where, despite frequent physical examination of the heart, their existence had not been even suspected; and, *vice versâ*, seen the pericardium of model health, where an adherent condition of the two laminæ had with overweening confidence been announced. Still, though difficult, the attempt must not be given up,—and the following considerations will help the observer materially.

The heart itself being either quite healthy, or not seriously changed in bulk or structure, the signs will vary, first, with the closeness of the pericardial adhesions themselves,—and secondly, with the absence or presence of pleuritic adhesions in front of the heart. So essential is the latter point, though ignored by writers generally on the subject, that it forms the ground of a distinction of the physical signs into two natural classes, as follows:—

PHYSICAL SIGNS OF PERICARDIAL ADHESION.

(*a*) *Pleuritic adhesions in front of the heart absent.*	(*b*) *Pleuritic adhesions in front of the heart present.*
Apex-beat in natural site; or retained above, in the spot it was raised to during the acute stages.	The same.
Apex-beat not notably lowered by inspiration.	This peculiarity still more marked.
Sometimes distinctly defined apex-beat wanting.	The same,—or it may recede with the systole.
Depression of præcordial region, about fifth to seventh left cartilages; or superiorly at the base of the heart.*	This more marked.
No dimpling of surface with cardiac action.	Epigastrium at left costal angle may dimple inwards with the ventricular

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* Case of Craddock, Clin. Lectures, Lancet, loc. cit.

	systole—especially if the heart and diaphragm be adherent.
Little or no undulating movement of heart.	Undulating movement more or less, sometimes excessively, well marked [47].
Respiration-movements slightly less active over heart than natural.	Respiration-movements almost null over heart.
Percussion-dulness about large vessels above third left cartilage from pericardial induration-matter.	This still more marked from the additional influence of pleural induration matter, and condensation of neighbouring lung-substance.
Pericardial clicking sounds about roots of vessels, or common friction-sound from loose adhesions,* or slight single rub with systole.†	The same.
Complete agglutination may exist without jogging, trembling motion of the heart.	Jogging trembling motion of the heart: (still, even here, this is rare, if the heart's bulk be natural).
Even though the two laminæ be agglutinated, the heart will fall from side to side as the patient's posture is similarly changed from right to left.	With such additional adhesions the heart will be fixed, and not moveable from side to side.

It has been said that feebleness, even to extinction of the second sound, is a sign of pericardial adhesion: I doubt whether the two things, when associated, have ever any direct connection; and I know that complete agglutination may coexist with a perfect second sound, and this, I may add, even though the wall of the heart be locally infiltrated with cancer.‡

Friedreich thinks that sudden diastolic collapse of the jugular veins, combined with depression of the entire supra-clavicular region, may prove to be a sign of pericardial adhesion.§ He admits, however, he had seen the association but once.

If new effusion occur in a formerly inflamed pericardium, the continuance of respiration in the præcordial region is a sign of adhesion, pericardial and adjacent pleuritic combined.‖ A past pericarditis does not prevent the characteristic signs of effusion occurring a second or a third time, provided there be not complete agglutination; friction-sound, percussion-dulness of triangular outline, and twisting of the heart's apex upwards may all be present with, and through, the new attack.

(*b*). AGGLUTINATION OF PERICARDIUM COMBINED WITH CARDIAC HYPERTROPHY.

396. In the second form, where agglutination of the serous

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* Kennedy, U. C. H., Males, vol. i., p. 67.

† Jones, U. C. H., Males, Feb. 1861; proved to have depended on an annular belt of calcification in the agglutinated laminæ: heart in U. C. Museum.

‡ Scammel, U. C. H., Males, vol. xvii., p. 276.

§ Hertzkrankheiten in Virchow's Handbuch, page 262, note. 1855.

‖ Craddock's case, Lancet, loc. cit. 1849.

surfaces and hypertrophy of the heart coexist, each condition modifies the signs of the other. Thus the heart's apex may beat as high as natural in spite of the enlargement; if, however, the hypertrophy be very great, the apex falls, for the material of agglutination stretches. An unusual tendency to extension of percussion-dulness upwards is observed. Systolic dimpling is very marked; the lower end of the sternum and the adjacent left cartilages may be drawn in at the left costal angle. Jogging, trembling action of the heart may be highly developed.

(*c*) AGGLUTINATION OF THE PERICARDIUM COMBINED WITH CARDIAC ATROPHY.

397. The cardiac atrophy, distinctive of the third form, comes of tight embrace by the material of adhesions; especially, in all probability, if the coronary arteries be accidentally pressed upon. But such atrophy is of excessively rare occurrence: a fact I ascribe to the rarity of thick strata of true induration-matter in chronic pericarditis.

The *physical signs* of simple adhesions will be conjoined with those of diminished size of the heart.

398. *Prognosis.*—(*a*) Adherent or agglutinated pericardium, so long as evidences of enlargement of the heart are deficient, is a condition perfectly compatible with healthy action of the heart, and has never been shown to be capable of destroying, or even distinctly shortening, life. That bodies are every now and then met with in the dead-house in which the pericardial sac must have been obliterated (as the history shows) for years, and where, nevertheless, death has been the result of some totally disconnected affection, is a fact so familiar as to need no special illustration.

(*b*) Where atrophy of the heart—a very rare result—arises from tight embrace of the material of adhesion, no very serious disturbance ensues. Here, again, there is failure of proof that life is jeopardised.

(*c*) When sequential dilated hypertrophy ensues (notably more usual on the left than the right side) the prognosis becomes greatly more serious, and just in proportion as the amount of dilatation exceeds that of hypertrophy. Life is probably not often prolonged beyond two to three years after overgrowth of the dilated species has become distinctly manifest symptomatically.

The practical questions then come to be: First, in what proportion of cases of agglutinated pericardium does increase of bulk follow; and, secondly, when such increase actually occurs, what average time is required for its accomplishment? Now, unfortunately, to neither of these questions can a trustworthy reply be given.[*] My own clinical records indicate that in about one-half the cases of adhesion, enlargement sooner or later follows; but I cannot supply even a vague notion of the average time in which the enlargement begins to assert itself *clinically*. A case will now and then occur in which the heart's outline and force of action seem, the former to widen, the latter to increase, almost coetaneously with completed agglutination [382].

In estimating the prognosis, cases where endocarditis has in addition damaged a valve, must of course be excluded.

399. *Treatment.*—In the three varieties of condition, just reviewed, the treatment in regard of the existing material of pericardial adhesion is identical. The absorption of that material may be encouraged by the use of ioduretted applications, by mercurial inunction, and by the internal administration of liquor potassæ and iodide and bromide of potassium. A course of the Woodhall or Kreuznach waters, internally and externally, is always worthy of trial.

If irritative action supervene, local depletion by a few leeches, blisters, caustic solution of iodine, and free purgation, are the chief remedies. Small doses of aconite and hydrocyanic acid will quiet irritable action. Subsequently the external use of belladonna and aconite will be found useful.

When dilated hypertrophy supervenes, the treatment must be essentially directed to that affection, on the principles elsewhere explained.

(*d*) CHRONIC PERICARDITIC EFFUSION.

400. The symptoms and signs of fluid stagnating, chronically, in the pericardial sac are very closely—minus the pyrexia—the same as those of the effusion-period of the acute disease.

401. If these signs continue unchanged by ordinary means of treatment (hydragogue purgatives have little effect), paracentesis

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[*] H. Kennedy, collecting 90 cases of adherent pericardium (apparently from museums and catalogues) finds the heart was healthy in 34, diseased in 56: purely hypertrophous in 25, hypertrophous and dilated in 26, atrophous in 5. Cases of valvular disease are purposely excluded; but this exclusion, though in one sense founded on correct views, would tend to lower the ratio of cases of diseased heart dependent on adhesion,—for it is impossible to deny that when the three things, pericardial adhesion, valvular impediment, and cardiac enlargement coexisted, the first of the three conditions may by possibility have played as important a part as the second in generating the third. (Edin. Med and Surg. Journal, vol. iii. 1857–58, p. 985.)

of the pericardium becomes justifiable, as an *ultima spes*, provided urgent suffocative symptoms exist. The patient is certainly not placed in a worse position by the operation than he was before it; the immediate relief is extreme, and a certain very small chance exists of at least temporary recovery. Many years ago Romero, of Huesca, published the results of eight cases, three without, five with, coexisting hydrothorax; in two of the former three the operation was successful.

Paracentesis should never be attempted, unless the exploring trochar have first given evidence of the presence of fluid—and of *non-loculated* fluid—within the sac. Even when fluid has positively and freely accumulated under observation we can have no certainty, until the exploring trochar is tried, that loculated strata of lymph may not impede its escape.

If the operation be determined on (the danger of wounding the mammary arteries, the larger pericardial vessels, and the great arterial trunks being borne constantly in mind), the integuments should be incised opposite the central part of the heart in the natural state of things, that is, at the upper angle of the fourth left interspace [11], or a little lower than this; a trochar should then be introduced cautiously into the distended sac perpendicularly to the surface, the patient being in the recumbent position with the head moderately low. Löbel, using an ounce glass syringe, armed with the needle of a hypodermic injection-syringe, operated in the third left, and twelve days later in the third right, interspace near the sternum. No injury was done to the heart: the patient died of double hydrothorax.*

The fluid, which escapes by jets, corresponding to the ventricular systoles, should be evacuated as completely as possible before the wound is dressed,—a syringe may even be employed to ensure this. But the orifice of the canula, before its removal, should occasionally be closed, lest too rapid abstraction of the fluid might produce evils of its own on the heart, accustomed as this has been, for a greater or less time, to considerable pressure. The practice of Romero, of allowing the fluid to gravitate into the pleural sac first, and thence outwards, seems unworthy of imitation.

Aran records a very remarkable case, in which he twice, within twelve days, tapped the pericardium, giving outlet on the first occasion to twenty-eight, and on the second to forty-nine, ounces of fluid. After the fluid had escaped, the operator on each occasion injected an ioduretted solution into the sac, on the plan which has sometimes proved curative of pleuritic effusion. As far as the operations were immediately concerned, the results seem to have been satisfactory.* See, also, Hæmopericardium [453], and Hydropericardium [463].

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* New Syd. Soc. Retrospect, 1871, p. 157.

e.—WHITE PATCHES OF THE PERICARDIUM.

402. A few words may here be added, as a sort of appendix to the subject of chronic pericarditis, concerning the so-called white patches of the pericardium.

403. These patches are of two kinds, perfectly distinct in their anatomical characters and in their mode of origin.

404. (*a*) In one variety these patches present themselves indiscriminately at all parts of the cardiac pericardium. Transparent at first, they eventually become opaque, white and tough, have well-defined margins, and are more or less readily separable from the serous membrane, which presents the appearances of health beneath them. They are evidently the result of an exudation-process on the pericardial surface, inflammatory in nature in the majority of cases: oftentimes acknowledged inflammatory conditions appear close by.

(*b*) In the other variety, the patch, scarcely ever met with except on the anterior surface of the heart, and principally of the right ventricle, has no sharp, well-defined margin, but is gradually lost in the surrounding pericardium, from which it cannot be separated by any manipulation. There is here a thickening of the proper substance of the serous membrane. The probability seems to be that they arise from long-continued friction, like corns from pressure, of the anterior surface of the heart.

405. The separable patch is rare,—the non-separable common. Bizot found the latter variety in 45 of 156 bodies. The same conscientious observer ascertained that this patch is of about three times more frequent occurrence in the male, than the female; and also that its appearance is notably under the influence of age: it is scarcely ever, if ever, seen before the age of seventeen, and does not become common until after forty.†

406. Little practical importance can be attached to either variety. If, however, the patch be at all prominent, and especially if it be rough on the surface, it may give rise to single

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* Med. Times and Gaz., 1856.

† Mém. de la Soc. Méd. d'Observation, t. i.

systolic friction-sound of maximum intensity in the lower sternal region.

II.—Endocarditis.

407. Endocarditis, like the inflammation of the outer membrane of the heart, occurs in the acute and chronic forms.

A.—ACUTE ENDOCARDITIS.

408. *Anatomical characters.*—(*a*) *In the membrane itself.* Here redness, florid in tint, sometimes distinctly vascular to the eye (depending, according to Luschka, on injection of the vessels in the deep elastic layer and connective tissue of the endocardium), may exist, pretty generally in patches, or sometimes more uniformly; or the hue may be pale dingy greyish yellow. The surface, deprived of its natural polish, feels more or less rough: the membrane becomes opaque, probably from filtration of exudation through the epithelium,—grows thick, soft, velvety, and can be stripped or scraped off in short patches; its tissue sometimes cracks, fibrinous coagula forming in the resulting fissures.

(*b*) *On the free surface.* In this position particles of exudation-matter, at first gelatiniform and transparent, subsequently opalescent, form thin strata which may be peeled off, or small oval elevations,—the latter either on the general surface or on that of the valves or amid the tendinous cords.

(*c*) *Under the endocardium* sero-fibrinous exudation-matter is deposited in small quantities.

(*d*) Accidental characters are rupture of the tendinous cords, especially of the mitral valve, which then curl up and act as the nuclei of fibrine-coagulation; fissuring of the semilunar valves, with deposits of exudation and of fibrine directly from the blood; altered shape of the valves; and softening with rapid destruction of their substance.

(*e*) Ulceration, or at least destructive softening of the endocardium, may be followed by extension of the process beneath; an opening has thus been effected between the auricles.

(*f*) Pus sometimes forms in the muscular tissue, immediately beneath the endocardium; upon its free surface, in fissures; and, it is alleged, in the substance of fibrinous coagula,—beyond all doubt softened fibrine has in the latter position, in some instances at least, been taken for pus.

(*g*) The formation of large coagula within the heart is frequent in severe cases; they bear all the acknowledged marks of production previously to death (the moment of their appearance can sometimes be fixed by physical signs during life), are sometimes the seat of recent vascularisation, and are commonly softened in the central parts.

(*h*) The endocardium of the left side is much more frequently inflamed than that of the right; the inflammation may, however, be limited to the right; affect both sides; or mere fractions of one. The valvular apparatus is more commonly and more strikingly involved than the general tract of the membrane. Endocarditis of the right side in the fœtus has been suggested as a probable cause of coarctation of the pulmonary orifice, and hence of non-closure of the foramen ovale.

(*i*) John Taylor[*] was, I believe, the first to show, as matter of observation, that the products of acute endocarditis, washed away with the blood, and impacted in distant capillaries, might lay the ground-work of pulmonary[†] hepatic, splenic, and renal secondary inflammations. This is a different achievement from demonstrating the similar embolic part played by particles of thrombi detached in chronic cases from the interior of the heart; embolism produced in this latter manner had been described long before his day, though he does not appear to have been aware of the fact [734].

In 300 cases of endocarditis, recently analysed by Sperling,[‡] embolism existed in 29 per 100 (on the right side in 3 per 100, on the left in 26 per 100) of the whole number: in right endocarditis the lungs were invariably the organs embolised. In left endocarditis the mitral valve was diseased in 88, the aortic in 49 per 100 of the cases.

The various organs were embolised in the following manner:—

Kidney	75 per 100
Spleen	51 ,,
Brain	20 ,,
Liver and Intestine	7 ,,
Skin	5 ,,
Medulla of Bones	3 ,,
Salivary Glands and Eye . . .	1 ,,

409. *Physical signs.*—(*a*) In acute inflammation of the endocardium, the heart's movement is seen and felt to influence the surface more extensively, more forcibly, and more abruptly than natural. The cardiac region is not bulged, nor is the point of the

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[*] Lectures in Lancet, 1845.

[†] Local gangrene of pulmonary tissue may follow such embolism. Vide Dis. of Lungs, 4th Ed., p. 385.

[‡] Inaugural Dissertation, Berlin, 1872, and Medical Record, January, 1873.

apex-beat, as in pericardial effusion, raised upwards; it may be even carried a little downwards and outwards. There is no undulatory movement, and no tactile thrill: Hope says he has observed the thrill of mitral regurgitation, but he gives no proof that the regurgitation was purely recent; I have not succeeded in finding thrill, when any certainty existed of the absence of old-standing mitral disease.

(*b*) The areæ of the heart's dulness, both superficial and deep-seated, undergo trifling increase: the former because greater energy of action brings the heart more uniformly forwards; the latter because the walls of the organ are turgid, and its cavities more or less clogged with blood. The area of dulness is never seriously increased, unless there be considerable distension of the heart by accumulated blood: then, mainly to the right.

(*c*) Auscultation discovers a murmur or murmurs, blowing in quality, soft and low-pitched. The murmurs of *purely acute* endocarditis may, as far as I have observed, be thus arranged in order of frequency: aortic obstructive; mitral regurgitant; aortic regurgitant; aortic obstructive and mitral regurgitant together; aortic obstructive and regurgitant together. Pulmonary systolic and diastolic murmurs are infinitely rare. I do not remember ever to have observed *acute* obstructive mitral murmur, nor *acute* regurgitant tricuspid murmur; the latter, especially, I believe to be at the least very rare,—a circumstance in accordance with the fact that most chronic tricuspid regurgitant murmurs are produced by simple incapacity of the valve to fill the widened orifice, without actual disease of its own tissue.

The site and rhythm of acute endocarditic murmurs, it is supposed, may change during the course of an attack;—lymph, it is presumed, may be absorbed, or washed away and deposited elsewhere, and a different species of murmur be consequently developed. I have not observed this in the *acute* disease; but I have known systolic aortic followed by diastolic aortic murmur, apparently from lesion of the valvular structure. The replacement of one murmur by another in chronic disease of the valves is spoken of elsewhere [119, 636].

The mechanism of the murmurs of acute endocarditis varies somewhat with the orifice concerned. But surface-roughness, fissuring, and lymph-deposits are the main causes of the obstructive class of murmurs; while those of regurgitant character may be traced to intertwined lymph interfering with the play of the tendinous cords, columnæ carneæ, and papillary muscles,—to actual destruction of the substance of valves,—or possibly in the instance of the aortic valves, and very surely in that of the mitral (*vide* Dynamic Intracardiac Murmurs, p. 88, *et seq.*), to a disordered dynamic condition of the apparatus concerned in closure. It is further conceivable, that friction of the blood over the general surface of the ventricular endocardium, roughened by lymph, may prove the efficient cause of murmur.

Such heart-sounds, as are not replaced by murmurs, present no constant character. Reduplication of the second at the base is common. Probably, at the outset, they are both intensified; and murmur-like prolongation of the first sound, before this actually gives place to true murmur, is sometimes noticeable both at the base and apex.

410. In cases where the *circulation through the heart's cavities is obstructed seriously*, either from accumulation of lymph and fibrinous coagula, or from rupture of a valve or chorda tendinea, the impulse becomes irregular in force and rhythm,—at first violent, subsequently feeble; the heart's dulness notably extends, especially to the right of the sternum; the sounds or pre-existing murmurs are enfeebled, or new murmurs may be generated. The suddenness of occurrence of these signs points to their source [742].

411. *Local Symptoms.*—The local symptoms of acute endocarditis are less varied and less severe than might be anticipated. Acute pain is rare, discomfort and uneasiness at the heart common; more or less subjective sense of palpitation exists; tenderness of the præcordial interspaces is at the least unusual, unless there be coexistent pericarditis [376].

412. *Systemic and Constitutional Symptoms.*—The symptoms belonging to this class vary with (1) the *free*, or (2) *obstructed* state of the circulation through the heart.

413. (1) In the *free* class of cases the decumbency is generally dorsal, the attitude quiet; but jactitation of the arms occurs in some instances. The temperature scarcely, as a rule, more than subpyrexial, may be high, or low to collapse-point (the concomitants and the effects of the disease regulating the heat more than itself). The integuments remain unchanged in colour; the joints are rheumatic, or unaffected; no special sensation of dyspnœa is complained of; the respiration holds its natural ratio to the pulse, so long as the orifices are not seriously obstructed, and no secondary pneumonia has occurred; sometimes a little dry cough exists without bronchial or other rhonchi: the pulse is not remarkably accelerated, ranging between 80 and 120;—Taylor's

statement that it loses in frequency at the outset of endocarditis, I have at last had an opportunity of confirming; but in what proportion of cases this initiatory slackening occurs, and whether essential or non-essential, remains to be determined. The blood is hyperinotic in the sthenic disease; if the inflammation be the effect of phlebitis, pyohæmia, &c., the clot is soft, but little, if at all, buffed; endocarditis may also, in all probability, *secondarily, cause the latter condition of the blood by the circulation of its own inflammation-products.* The urine is simply febrile. Cephalalgia exists commonly more or less,—slight wandering may occur at night, but otherwise the head remains free. I once saw acute mania occur during the convalescence of endocarditis, of rheumatic origin, and unaccompanied with pericarditis.* Choreal, or other reflex, symptoms are not induced, if the disease remain simple.

414. (2) In the *thrombal* and *obstructive* class of cases, the action of the heart, suddenly at the moment of obstruction, becomes excessively frequent, uneven, and irregular; the pulse small, weak, irregular in force and rhythm, mounts to 130, 140, 160, or even more. Semi-syncope, pallor, coldness of surface, anxiety, and jactitation, inclination to orthopnœa (which the patient resists from its increasing faintness), with, eventually, the symptoms of more or less complete suspension of pulmonary circulation (subjective and objective), its breathlessness from want of blood to oxygenise,—lividity of surface, turgescence of the face and neck, prominence of the eyeballs, puffiness of the ankles, supervene,—the brain suffers also congestively, as exhibited by fitful snatches of sleep, convulsions, delirium, and somnolence, lapsing into fatal coma. I have seen these symptoms in a minor degree, and passingly, in certain cases of endocarditis, which terminated favourably,—in all probability, in those instances, small thrombi had formed, and subsequently undergone disintegration and solution. The symptoms of rupture of a chorda tendinea during the acute disease are extremely similar; the effects on the cardiac circulation must, indeed, be closely analogous.

The blood, in certain cases of endocarditis, receiving the products of the inflammation, undergoes alteration of composition. Rigors, heat of surface, and profuse perspiration recurring irregularly, dull, earthy, yellow discoloration of the skin, but not of the conjunctiva, diarrhœa, more or less bilious, pinched, anxious countenance, intense prostration, and muttering delirium, subsequently cool clammy skin, and blueness of the finger-ends, announce this occurrence; and are followed by, or associated with, the evidences of secondary nodular pneumonia or hepatitis.

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* Cooper, U. C. H., Males, vol. i. p. 129.

415. *Course.*—Rheumatic endocarditis may run a perfectly *latent* course in regard of local and general subjective symptoms.

416. *Causes and conditions of origin.*—Co-arising with endocarditis, or generated at least by the same influences, may be found rheumatic arthritis, pericarditis, myocarditis, aortitis, pneumonia and pleurisy. Aortitis, whether præcursory, synchronous or sequential, is a much rarer attendant, at least as far as proof goes, than might be expected. "Bright's disease," rheumatism, and pyohæmia, are the main diathetic and acute specific affections, that clearly promote the occurrence of endocarditis. Neighbouring inflammations, old cardiac disease, injuries to the præcordial region and violent efforts are sometimes traceable as its causes. The existence of old valvular disease renders the endocardium more prone to acute inflammation.

417. *Diagnosis.*—The diagnosis of acute endocarditis is essentially based on the existence of the general aspect of acute disease, cardiac uneasiness, excited action of the heart, and endocardial murmur,—this murmur occurring in a person presumedly free from prior endocardial disease, and presenting no other obvious affection to explain the acute character of the existing illness.

But suppose an endocardial murmur is detected on the first examination of a patient, labouring obviously under some acute attack, and concerning whose past cardiac history no trustworthy information is to be had, that murmur may obviously be *new*, or it may be *old*. Now, granting that it is new, it may not be the product of endocarditis. For the excitement of the heart may depend on some other, latent, inflammation or as yet unevolved disease; and this excitement, coupled with a modified state of the blood, may suffice to generate a murmur. Thus, occasionally, at the outset of pneumonia, of the exanthemata, and of any inflammatory state, in persons whose blood chances to be in the least spanæmic, murmur occurs. But such murmur, purely of blood-origin, is necessarily systolic and basic; if there be new murmur either systolic or diastolic (præsystolic) at the apex, or diastolic at the base, a complication of acute endocarditis must be admitted; if the murmur be basic and systolic only, it may be hæmic, or it may be endocarditic, and consequently the diagnosis must be deferred. Besides, deposition of fibrine may take place, as an acute process, amid the cords, or at the edges of the valves, and induce murmur, without the intervention of inflammation.

So far the murmur has, for argument's sake, been admitted to be new; unfortunately the great difficulty is often to determine that it *is* new. Now two cases present themselves here: (*a*) the murmur exists, when the patient is first seen; or (*b*) it is developed, after observation of him has commenced.

(*a*) A murmur being already present, the circumstances *within itself*, favourable to recency of origin, are softness of blowing quality, lowness of pitch, systolic rhythm and aortic constrictive or mitral regurgitant mechanism. The circumstances hostile to recent origin are roughness of quality, high pitch, diastolic rhythm (indeed this is in all probability absolutely conclusive, unless there be systolic murmur at the same orifice), and seat at the tricuspid orifice; direct mitral (or præsystolic) left apex murmur, also, I believe, is never discoverable at the early stage of fresh inflammation. The presence or absence of affections, with which endocarditis is commonly associated, furnishes a guide not to be despised; but without caution the observer may readily be led into error, as in acute rheumatism, by too implicit trust in this very guide. The condition of the pulse cannot be confided in for diagnosis; nor does the thermometer afford any trustworthy sign.

(*b*) An endocardial murmur, *developed under observation*, *at the early period of an acute attack*, is almost a sure index of endocarditis; but even here there are sources of fallacy. In the first place appear those just enumerated, on the hypothesis of the murmur being by admission new, and yet of hæmic origin. In the second place, general collapse and failure of the heart's power may, on the first examination of a patient, prevent a given murmur, of which the physical chronic conditions exist in perfection, from being heard. Reaction takes place, and a murmur becomes audible; that murmur may be chronic murmur solely, or it may be an acute and a chronic murmur combined; but it is *not* that, which it would alone seem to be, namely, an acute murmur *solely*.* Again, to have value as a positive sign of recent endocarditis, the murmur must be developed at an *early* period of acute disease; if towards the close, it is almost invariably systolic in time, basic in seat, and a consequence of spanæmia. Further, the possible occurrence of murmurs of dynamic mechanism, muscular and valvular, must not be forgotten. Not only in chorea, but in other nervous diseases, and in nervous conditions supervening in the course of acute maladies in general, is their occur-

* Kernis, U. C. H., Females, vol. ii., p. 237. The statements in the text are made on clinical and *post-mortem* evidence.

rence a possibility, nay a probability. Such dynamic murmurs are generally systolic and seated at the apex; but there is much reason to conjecture that murmurs may also occur dynamically at the base from perverted action of the sygmoid valves [*vide* Dynamic Murmurs, p. 91].

If, during the course of an acute disease, endocardial murmur assumes fresh sites, this is a very strong, though not an absolute, sign of its dependence on recent inflammation; and, admitting its value, such change is, we have seen, at the least very rare. Again, if a murmur of a certain site and rhythm disappear and return within a short period, the changes might seem proof positive of recency of the cause of the original murmur; but they are not so, at least of murmurs of all varieties of localisation. Thus, I have known a direct mitral murmur, essentially organic and attended with great constriction of the orifice, come and go from one day to another: some dynamic conditions must now have obstructed, now have promoted, the occurrence of the murmur [p. 101].

And there is yet another possible cause of error: an endocardial murmur sometimes occurs, as originally mentioned by Latham and Stokes, and as now generally admitted, shortly before death, from the impediment of coagulated blood. Fortunately this *præ-mortem* murmur is of rare occurrence: how, if it at all, it may be distinguished from an endocarditic one, remains to be ascertained. In point of fact even the coexistence of undoubted signs of intra-cardiac thrombosis would not prove the murmur to be non-endocarditic.

So far intra-cardiac murmur has been assumed to be unfailingly present. But may it not be absent in acute endocarditis? The conditions producing murmur are roughness of valves, lymph among the tendinous cords, insufficiency of valves from puckering, and notable roughness, from lymph, of the ventricular surface: now as endocarditis may exist without any one of these anatomical effects, the inference is unavoidable, that it may occur without murmur. I have seen distinct patchy redness with thin films of lymph on the ventricular endocardium, where there had been recent excitement of the heart without newly developed murmur.*

Endocarditis will be distinguishable from pericarditis by the less amount of pain, by uneasiness not being increased by a deep breath, cough or change of posture, by absence of præcordial tenderness, by less amount of laboured breathing, and by the general difference of the physical signs. The distinctions of endocardial and pericardial murmurs, and the alleged value of stethoscopic pressure as an aid in their diagnosis, are elsewhere considered at length [133, 134].

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* "Shreddy lymph on surface of left columnæ carneæ recent," without change in character of pre-existing chronic murmurs. Kernis, U. C. H., Females, vol. ii., p. :44, March, 1847.

The occurrence of thrombosis within the right heart, with actual formation of more or less solid coagula, may be very strongly suspected if, with the *sudden* appearance of the symptoms enumerated above, a notable increase in the area of percussion-dulness be detected. But rupture of a sygmoid valve, or of a tendinous cord, will produce closely the same effects.

418. *Prognosis.*—Considerable difference of opinion, not only in regard of minor but of major points, may be traced in the statements of authors concerning the ultimate effects of endocarditis. The results, that have fallen under my own observation, may be set down as follows:

(1) Very serious valvular disease, followed by implication of the heart's substance, and all their combined consequences: the ensuing affections of the heart are, in their order of frequency, eccentric hypertrophy, simple hypertrophy, simple dilatation, and, in infinitely rare cases, eccentric dilatation.*

(2) Slight valvular disease, with habitual palpitation.

(3) Slight valvular disease, with palpitation under excitement.

(4) Simple murmurs, without any positive cardiac functional disturbance; no morbid palpitation occurring even under severe exercise. This is the most favourable result that I have actually met with. *An indubitable endocarditic murmur, holding on from the beginning to the end of the acute disease, never, so far as I have known, totally disappears in after-life.* I am aware that in this matter my experience is not precisely in accordance with certain printed statements,—the correctness of which I do not for a moment question. I merely affirm what my own observation has taught me. Still it must be admitted there are some possible sources of mistake in this matter, the principal of which appear to be the following. Temporary obscurity of a recent mitral murmur sometimes arises from the weakness of the heart attending convalescence, and leads to the idea that the murmur is gone, or will go, completely: as the general vigour improves, the murmur recovers its distinctness; but meanwhile the patient has been

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* The relationship between the part of the heart affected and the diseased orifice will be considered with valvular affections generally; for the endocarditic origin of the valve-disease gives no *special* character in this way.

lost sight of. Again, a mistaken inference is sometimes drawn as to the disappearance of murmurs, from their point of maximum intensity undergoing change. This is especially true of the murmur of mitral reflux, which sometimes inexplicably becomes inaudible at the apex and appears in full vigour at the axilla. And, further, a murmur of anæmia, arising in the advanced period of the disease, is sometimes mistaken for one of really inflammatory mechanism.

(5) On the other hand, death is rare from acute endocarditis alone; still, the disease does occasionally kill, both by secondary impregnation of the blood, by serious thrombal obstruction of the intra-cardiac circulation, and by embolism.

419. *Treatment* (*a*).—The ultimate dangers of acute endocarditis are extreme; they involve the perils of various forms of valvular disease, combined with altered dynamism and oftentimes changed statical conditions, both of capacity and of substance, of the heart itself. Acute endocarditis is then a disease in which it is in the highest degree desirable to promptly exercise a controlling power, if we possess this. But, I confess, I have never had satisfactory proof, either from observation or reading, that lavish blood-letting and mercurialisation really stay the course, or remove the proceeds, of the inflammation-process in this locality; while at the same time no other agencies have even been asserted to possess the powers in question. I believe, consequently, if the direct and instant suspension of acute endocarditis by art be a possible feat, the means of accomplishing it are reserved for future discovery.

Meanwhile it appears reasonable the course of treatment should be that recommended for acute pericarditis.

(*b*) If there be proven tendency to, or even if the circumstances merely suggest the likelihood of, thrombosis within the right heart, ammonia should be freely given. The physiological researches of Richardson explain, while they demonstrate, the utility of the dissolvent method,—besides the stimulus of the ammonia is likewise of essential aid. Brandy may be required every hour or half-hour, in quantities measured by the feebleness of the pulse and degree of general exhaustion. I have seen relief obtained by cupping over the heart: but the patient's strength is rarely, when such obstruction occurs, in a condition to bear the loss of even an ounce or two of blood,—the relief was but temporary, and the practice does not appear to be deserving of imitation. The application of Junod's dry-cupping apparatus to the lower extremities is a safer measure.

(*c*) When the affection seems lapsing into the chronic state, iodide of potassium and liquor potassæ, with bitter tonics, become the best remedies; and iodine-inunctions over the cardiac region appear occasionally useful.

B.—CHRONIC ENDOCARDITIS.

420. Chronic endocarditis is anatomically signified by thickening and opacity of the membrane,* uniformly or patchwise,—the surface being perfectly smooth or slightly and irregularly puckered. Successive strata, thin, opaque, faintly yellow, and filmy, may be peeled off from the free surface; or minute masses of hard, elevated, yellowish induration-matter separated with the point of the scalpel, leaving the endocardium, apparently uninjured, beneath.

The valves suffer variously. Thickening by infiltrated induration-matter, with hardness, opacity, and puckering of their substance, sometimes associated with diminished depth from acute destruction or chronic contraction, are the most common appearances. Calcification, or pseudo-ossification, may ensue in patches, nodules, or spiculæ. Adhesion of the divisions of a valve to each other, or to the neighbouring arterial or endocardial surface, is sometimes seen: the former, most common in the aortic valves, may cause them to pouch in the wrong direction; but such adhesion, converting them into a single indurated ring, occurs to perfection in the mitral valves also. Thickening with shortening of the mitral cords, sometimes conjoined with special thickening of the endocardium of the papillary muscles and cirrhosis of these muscles themselves, is clinically one of the most important of chronic changes.

Warty fibrinous products, polypoid or sessile, soft, firm, or calcified, form on the valvular surfaces exposed to the blood-current. The free edge of the mitral, less frequently of the tricupsid valve, frequently presents a semi-transparent beaded thickening, which is not clearly inflammatory, or even morbid.

421. *Symptoms.*—Although, very possibly, certain uneasy sensations about the heart [263] and proneness to palpitation, may depend upon chronic changes in the ventricular or auricular endocardium, yet there is no surety of knowledge on the point. Hence, in point of fact, chronic endocarditis is solely known clinically by its effects on the valves and orifices of the heart; and there is nothing in the statical or dynamic characters of valve-disease originating in inflammation, distinguishing it from that produced

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* The student must not forget the natural excess of opacity and thickness, particularly as age advances, in the endocardium of the left auricle.

by other causes. For the clinical history of chronic endocarditis the reader is consequently referred to the section on Valvular Affections in general.

III.—Endo-Pericarditis.

422. The combination of the two inflammations, as each separately, may exist in the acute and chronic forms.

ACUTE ENDO-PERICARDITIS.

423. Acute inflammation in a certain proportion of cases attacks both membranes of the heart,—either, to all appearance, coetaneously or consecutively.

424. The relative frequency of the double inflammation, and of the two singly, has been variously estimated by different persons. Rheumatic endo-pericarditis is, if Latham's returns be accepted, somewhat more frequent than pericarditis alone, greatly less frequent than endocarditis. But it is difficult to resist the conviction that the great excess of endocarditic cases in part depended on old intra-cardial murmurs being classed as new; especially as the results of other observers do not tally with those of Latham.

There are two methods of determining the question of frequency, here started,—the clinical and the anatomical. Now both have their difficulties. Thus, in respect of the clinical method, it is certain, that pericarditic friction-sound often renders the detection of existing endocardial murmur difficult or impossible; and that a large amount of pericardial fluid also commonly weakens endocardial murmur, if present; while the pressure of such fluid may, very possibly, in some rare instances, create an obstructive intra-cardiac murmur quite independently of endocardial inflammation [135]. The signs and effects of endocarditis on the other hand cause no difficulty in the discovery of pericarditic signs. In other words, pericarditis obviously existing, endocarditis may very well be present, though its signs are imperfectly discoverable; whereas the converse does not hold good.

So far, then, as these facts go, the tendency of clinical observation would be to under-estimate the relative frequency of endocarditis. Besides, as I have already mentioned, acute endocarditis, anatomically demonstrated, does not of necessity produce murmur. But, on the other hand, the similarity of old structural, as well as of certain dynamic and anæmic intra-cardiac murmurs, to those freshly endocarditic, helps, though in a fallacious fashion, to balance the account.

In respect of the anatomical method, endocardial thickenings have sometimes, though purely hypertrophous, passed for inflammatory. *Vice-versâ*, I have strongly suspected in some few instances that endocarditis had really existed, but all congestive state had disappeared before death, and any exudation-products been washed away by the blood-current.

Making such allowance, as I have been able, for these conflicting sources of embarrassment, I find my hospital-experience, both in acute rheumatism and in "Bright's disease," gives the first place in frequency to pericarditis, the second to endo-pericarditis, and the last, *longo intervallo*, to endocarditis alone.

425. *Prognosis.*—The *immediate* prognosis of the double inflammation is worse, but not very seriously worse, than of pericarditis alone,—materially worse than of endocarditis alone. The *ultimate* prognosis of the combined inflammations is very sensibly more serious than of either separately. As we have already seen, it is coexistent valvular mischief that seems mainly to entail the evils assigned by some writers to pericardial adhesions.

CHRONIC ENDO-PERICARDITIS.

426. The clinical history of this state, in its practical aspects, will be found under the head of Valvular Diseases.

IV.—Carditis or Myo-carditis.

427. Inflammation of the muscular structure of the heart, like that of the membranes, is met with in the condition of an acute and of a chronic disease.

ACUTE CARDITIS.

428. Acute carditis presents itself under four forms or conditions, sufficiently different from each other to require separate consideration.

429. *First Form.—Acute carditis, as an attendant on endo-pericarditis.* The anatomical characters of this form of the disease are well known; they are of tolerably frequent occurrence, on a limited scale, in the strata of fibres nearest the inflamed membranes, especially the pericardium. I have seen exudation-corpuscles and pus-cells amid the fibres under these circumstances.

430. *Symptoms.*—But, clinically, the effects of such carditis are not understood; whatever they are, they are lost in the more striking phenomena of the membranous inflammations. Possibly great weakness and fluttering character of the pulse may some-

times be due to inflammatory softening of the left ventricle, in cases of pericarditis without much exudation or fluid effusion, and therefore without an efficient mechanical cause of obstructed action. But, although this view of an intrinsic muscular failure has been adopted by Stokes, it would apparently prove immensely difficult to show satisfactorily that the weakened action does not really arise out of the influence, either direct or reflex, exercised by the inflammation on the nerves of the heart (*vide* Cardiac Paralysis) [268—272]. And a further ground on which I raise this objection is, that in certain indubitable cases of limited purulent infiltration of the heart, the action of the organ has not been found at the outset of the attack either notably deficient in vigour, or seriously irregular in rhythm. I of course refer to cases wherein the absence of acute pericarditis is positively averred by their narrators.

431. *Second Form.—Idiopathic and primary general inflammation of the muscular and connective tissues of the heart.*—This is an affection, to say the least, of extreme rarity; nor is it certain any evidence exists of its ever occurring as an independent and isolated disease.

432. *Anatomical characters.* — Vascular injection, deepened colour, with crispness, not real firmness, of the texture, seem the earliest alterations. Exudation, sero-sanguineous, or of lymph, or of pus, either in the form of infiltration or abscess, ensues; the texture softens even to pulpiness, and, commonly of pale yellowish, greyish, or greyish-brown hue, may, if there be much infiltration of blood, acquire a blackish tint. If there be local softening and abscess, the part affected may bulge outwards, form a variety of true cardiac aneurism, and actually give way into the pericardium. Ulceration extending a certain depth into the wall of a ventricle, or perforating either the pericardium or the ventricular or auricular septum, has rarely, gangrene still more rarely, been seen. The left ventricle is the part most frequently affected.

The pericardium seems almost always to undergo secondary implication; the endocardium and valves escape,—a fact the more strange, as there is strong ground for believing that myo-carditis originates in some special poisoned state of the blood, and is never truly a primary idiopathic disease.

433. *Causes.*—The narratives of the few cases of general carditis on record furnish no clue, at least of trustworthy character, to its ætiology. Of the influence of age nothing is known; it is probably insignificant, for the disease has been seen in boys, and in men

aged upwards of seventy. Hitherto the male sex has furnished almost every recorded case.

434. *Symptoms.*—The clinical history of general carditis is at present singularly imperfect. The rarity of the affection, and its constant association with other grave disease, acute or chronic, or both, serve to explain this imperfection. The only local symptoms appear to have been weakness and irregularity of action and cardiac distress, without positive pain (sometimes even without uneasiness) in the præcordial region, unless where pericarditis coexisted. Pyrexia of adynamic character, with a pulse rapidly becoming feeble and irregular, pains in the limbs (taken for signs of rheumatism, though the joints were free), rapidly ensuing coldness of surface, anxiety of countenance, lividity, and jactitation have indicated the gravity of the affection.

435. *Physical signs.*—Of the physical signs little can be said. There is no reason why the percussion-results should deviate from the normal standard; and in the muscular inflammation itself there exists no efficient cause of intra-cardiac murmur. Impulse excited at first, quickly growing feeble (not necessarily irregular in rhythm, or unequal in force), is in point of fact the only altered physical condition theoretically to be anticipated.

436. *Co-existences.*—In the majority of cases the heart-disease has been thrown into the shade by the predominance of cerebral and other symptoms (cephalalgia, delirium, convulsions, and coma), indicating some form of poisoning of the blood, allied to or identical with pyohæmia. In a few cases (for instance, that of Burrows*) recent pustulation of the skin, apparently secondary, and in all probability due to capillary embolism, has been noticed.

437. *Duration.*—Calculated from the moment of supervention of local symptoms, death has occurred at periods varying between a few hours and a week.

438. *Diagnosis.*—I am not aware that the existence of carditis has ever been diagnosed; nay more, it would appear that the symptoms, the affection has really induced, have, during life, been referred, to the satisfaction of the observer, to some other state which post-mortem examination did, or did not, prove to be present.† I have no personal experience of the disease: and can only suggest the following *à priori* guide to its detection. If

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* Medical Times, Dec. 1853.

† Thus in the oft-quoted case by Stanley (Med. Chir. Trans. vol. vii.), all who saw the patient thought he had disease of the brain or membranes, which proved to be healthy.

grave general symptoms of adynamic pyrexial character coexisted with a state of heart's action, at first excited, and then rapidly losing power, pains in the limbs and cerebral symptoms, while the absence of endocarditis, pericarditis, typhoid (Peyerian) fever, and other adynamic pyrexiæ, could be made matters of demonstration, a fair suspicion, but nothing more than this, might arise of the existence of carditis, especially if the patient were a male.

439. *Prognosis.*—It seems scarcely worth while to speak of prognosis, where diagnosis is unattainable. Could, however, such diagnosis be obtained, it would be in the present state of knowledge equivalent to a verdict of death.

440. *Third Form.—Partial carditis.*—Partial carditis sometimes occurs, producing abscess, ulceration, and rarely actual gangrene; but of these states no positive clinical signs are known. If perforation or rupture occur, as they sometimes do, the symptoms will vary with its direction.

441. Abscess of the heart, a not very uncommon attendant on pyohæmia, is excessively rare as an independent state. It has, however, been observed in youth and in advanced age,—in the male sex.

442. *Symptoms.*—There may be a total absence of symptoms to direct attention to the heart, even under the gravest conditions,—as, for instance, where the abscess communicated with the pericardium and interior of the left auricle.* In another case, where the abscess in the wall of the left ventricle had burst into its cavity and that of the pericardium, the patient, a boy aged thirteen, was suddenly seized with vomiting, followed by collapse and coma, and died the next day: nothing is said appertaining to the heart by the narrator, except that the pulse was rapid and indistinct, and the sufferer faint.†

In another case, that of a male, aged sixty, the patient, suddenly seized while at work with "coma, *cyanosis*, and great prostration," died calmly on the fourth day.‡ There was no reopening of the foramen ovale, nor (as may be inferred) perforation of the ventricular septum. When perforation of that septum has occurred, cyanosis has been known to follow; while, it is affirmed by Bouilland, neither blue discoloration nor any other serious symptom may arise to announce the occurrence. Rupture of the heart into the pericardium commonly produces the gravest effects, as will further on be fully shown.

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* Howitt, Provincial Transactions, vol. xv., 1847, p. 193.

† Chance, ibid., p. 113.

‡ Stallard, ibid., p. 105.

443. *Mode of origin.*—It seems probable enough, as contended by Stallard (*loc. cit.*), that abscesses of the present kind may sometimes follow on local hæmorrhage.

444. *Diagnosis.*—This affection lies at the present day beyond the pale of possible diagnosis.

Fourth Form.—Secondary cardiac abscesses.—Minute collections of pus form in the heart's substance in a certain proportion of cases of pyohæmia and phlebitis,—greatly less frequently however than in the lungs and liver. Here, as in other organs, these so-called abscesses are often really composed of liquefied exudation-matter alone. The symptoms are yet to be worked out.

CHRONIC CARDITIS.

445. Shortening with thickening of the papillary muscles and columnæ carneæ, and infiltration of their substance with induration-matter, due to a chronic low congestive process, mainly derives interest from its interference with the closure of the mitral, in very rare instances of the tricuspid, valves. There is no character in either a mitral or tricuspid regurgitant murmur, distinctive of this special mechanism.

446. The more important bearings of chronic inflammatory change will be considered under the heading of CIRRHOSIS OF THE HEART.

§ V.—CARDIAC HÆMORRHAGES.

I.—INTRA-MUSCULAR HÆMORRHAGES.

447. Circumscribed extravasations of blood into the muscular substance occur to very various amounts. The fluid may form mere specks, or apoplectiform nodules as large as a walnut. Or portions of tissue may be infiltrated with blood: the implicated muscular substance is then softer than natural; and, as deficiency of consistence may be traced beyond the limits of the infiltrated part, it seems obvious that a softening-process takes the lead in the morbid changes. This softening involves the minute vessels, —hence the extravasation.

448. I am uncertain whether this form of local softening of the heart with blood-infiltration ever lays the groundwork of true sacculated aneurism; but there is no doubt rupture or perforation of the ventricular wall may ensue. In both cases escape of blood into the pericardium follows: if the opening be at all large, instantaneous death is the necessary result; but if the communi-

cation be small and sinuous, the blood may make its way outwards as it were *guttatim*, and forming a layer of coagulum on the surface, temporarily close the external opening.

449. *Symptoms.* — Distinctive symptoms of intra-muscular hæmorrhage remain to be discovered, —if, indeed, any such really exist.

An individual previously known to have a weak, and not enlarged, heart, and to be free from aneurism, if instantaneously cut off by syncope and presenting *post mortem* the percussion-signs of accumulation in the pericardium, might be suspected to have perished from hæmorrhagic rupture of a ventricle; but obviously such mere suspicion would be worth nothing in a medico-legal point of view, and complete examination of the body would be imperatively called for.

II.—HÆMOPERICARDIUM.

450. (*a*) *Inflammatory.*—The effusion of inflammation is sometimes so much stained with blood, as to entitle the disease to the name of hæmorrhagic pericarditis. It seems probable that such escape of the blood-disks does not take place from the vessels, unless the constitution of the blood itself be affected. I have seen this variety of inflammation well marked in pyohæmia. But no signs or symptoms are known, whereby the hæmorrhagic addition to the effusion might be recognised during life. Neither is there any evidence to show that, in the event of recovery, the material of adhesion will be of different character from that observed in the more ordinary class of cases. The quantity of blood is sometimes very considerable,—quite enough to give a deep red colour to all the fluid in the sac: were paracentesis of the pericardium performed in such a case, the operator would very probably be led, for a moment, to believe that he had punctured some important vessel.

(*b*) *Traumatic or perforative.*—Hæmopericardium may be caused by wounds or by ruptures of the heart itself, by rupture of an aneurism of the aorta or of the heart, by rupture of a coronary artery or vein, by the giving way of cancerous substance, and possibly by filtration from a vessel within the pericardial tissues themselves. In all these cases the result is almost instantaneous death, from mechanical obstruction of the heart's action. Some of the series will again be referred to; the rest are devoid of clinical interest.

(*c*) *Scorbutic.*—The pericardium is pretty frequently the seat of extravasation of blood in cases of scurvy,—especially in some particular regions of the world. Thus in certain parts of Russia, scorbutic hæmopericardium seems as thoroughly endemic as hæmaturia in the Mauritius.

451. *Symptoms.*—The effusion of blood into the pericardial sac occurs with or without previous scorbutic symptoms, and the attack may be sudden, or so gradual that attention is scarcely drawn to the heart. Præcordial oppression, without pain, or tenderness, and great dyspnœa, seem to constitute the main symptoms; the physical signs will, of course, be those of a pericardium distended with fluid.

452. *Terminations.*—The more frequent termination is by death; but recovery sometimes takes place by absorption of the blood,—the anatomical conditions remaining being very assimilable to those of chronic pericarditis. Indeed, it seems highly probable that the disease is from the first sub-inflammatory.

453. *Treatment.*—The treatment, locally, is by cupping,—constitutionally, that of the blood-disease present. In the majority of a number of cases, in which paracentesis was performed by Russian physicians, the ultimate result was unfavourable; but in all the immediate relief was extreme,—the patient seemed temporarily endowed with new life: and two cases are given of complete recovery. In one of these, related by Karawajew, three pints and a half of fluid are stated to have been removed from the pericardium; pneumo-pericardium followed: five months after the operation the patient was considered well.*

III.—Hæmo-endocardium.

454. Extravasations of blood under the endocardium in points, maculæ or actual ecchymoses, are by no means excessively rare. They sometimes occur to a notable extent in cases of protracted obstruction to the movement of the blood through the right cavities; and constitute one of the anatomical sequelæ of congestion of the heart's substance [363]. Nor can there be any doubt that these minute hæmorrhages play a certain, though not easily definable, part in working out the ultimate effects of muscular congestion.

455. Clinically this matter is a blank: there are neither known symptoms nor signs of this form of hæmorrhage.

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* British and Foreign Medical Review, July, 1841.

§ VI.—ALTERATIONS OF SECRETION.

I.—Œdema of the Heart.

456. I have seen the heart's substance of watery look, and distinctly infiltrated with serosity, both in cases of chronic pericarditis of the persistent effusion-form [400], and in cases of general dropsy. Nevertheless, ill provided, as it is, with connective tissue, the heart rarely becomes œdematous to any extent. In chronic pericarditis, it is rather the intervening texture between the heart and its inner and outer membranes, than its own substance, that undergoes infiltration.

457. In the present state of knowledge these conditions are merely anatomically interesting. Yet it seems probable that œdema, carried to any amount, may interfere with the heart's contractions.

458. Serous infiltration under the endocardium and between the layers of the sygmoid and of the mitral valves has also fallen under my notice; but I know nothing of either state clinically. It seems quite certain that such a condition of the sygmoid valves must alter the quality and tone of the second sound; whether infiltration can ever reach such a degree as to seriously impede the closure of the valves remains to be determined.

II.—Hydropericardium.

459. Hydropericardium, or dropsy of the pericardium, may be of *active*, *passive*, or *mechanical* origin,—or prove of *doubtful* mechanism.

(*a*) Active hydropericardium is very rare: I have, however, in some instances of "Bright's disease," known the pericardium fill with fluid,—the symptoms indicating an irritative state, while the signs (and hence, I assume, the anatomical conditions) of pericarditis were wanting. I once saw a case which suggested to my mind the question, whether, in true hydropericarditis, the plastic material might not be completely absorbed, and the serosity left behind,—constituting a sort of sequential active hydropericardium. But I have no positive answer to supply; and, possibly, the case referred to was one of active dropsy alone from the first.

When hydropericardium is active, it may be the sole dropsy in the body.

(*b*) Passive hydropericardium occurs as a phenomenon of general dropsy,—very rarely unless double hydrothorax be already present. The quantity of fluid (colourless, straw-coloured, or slightly blood-stained, but without lymph) is generally moderate,—from eight to twelve ounces; I have never seen more than the latter quantity.[*]

(*c*) Mechanical hydropericardium has, in some very rare cases, been traced to pressure of aortic aneurism and carcinoma on the great veins, and to morbid states of the coronary veins.

(*d*) The mechanism of hydropericardium is sometimes very difficult of explanation. Thus in a phthisical male, aged twenty-four, cut off in about four hours by perforation of the right pleura and pneumothorax, with little pain, but intense dyspnœa and general distress, the pericardium, pyramidally distended up to the second rib, was found to contain nine ounces and a half of clear faintly yellowish serosity, of specific gravity 1013, neutral reaction, and giving one-third albumen on boiling: there was not the slightest vascularity of, or lymph upon, the serous membrane, which simply looked macerated.[†] The lung was flattened against the side, but sufficient time had not elapsed for the occurrence of pleuritic changes. Could this hydropericardium have been the result of the sudden pressure on, and obstructed circulation in, the right lung with its sequential obstruction in the right heart and coronary veins?[‡] It is true, hydropericardium does not appear in the narratives of cases of phthisical perforation; but death rarely takes place so rapidly as this: in truth I have neither met with, nor heard of, a strictly parallel instance. Now, in cases of rapidly fatal type, yet of the ordinary class, where life is preserved for at least thirty-six or forty-eight hours, the first shock of the mechanical difficulty in the course of the cardiac circulation has had time to pass away, absorption has had time to commence, and may, before death, have removed, or nearly removed, the effused serous fluid.

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[*] A case recorded by Corvisart (Maladies du Cœur, 2ème édit. p. 52), where "about four pints, or eight pounds, of clear greenish serosity" were found in the pericardium, seems to have been one of chronic pericarditis. The distension which the sac must have undergone is the more remarkable, as the membrane is stated to have been thickened.

[†] Imlach, U. C. H. Males, vol. viii. p. 402.

[‡] This view receives strong support from a case of extensive effusion into the pericardium, caused by a fibrinous clot occluding the orifice of the coronary veins. Ralfe, Br. Journal, 1870, vol. ii. p. 553.

460. *Local Symptoms.*—Hydropericardium, however originating, has few subjective symptoms: acute pain and tenderness are altogether, and palpitation commonly, wanting; sensations of weight and oppression are alone complained of.

461. *Physical Signs.*—The physical signs are in the main those of hydropericarditis. But there is no friction-sound, no præcordial bulging, and the apex of the triangular dulness (mainly because there is less fluid, but partly because there is no inflammatory relaxation and rarefaction of the sero-fibrous membrane) does not mount so high as in many cases of pericarditis. The visible impulse may be very perfectly undulatory.

462. *Systemic Symptoms.*—The general symptoms are those of the disease on which the local dropsy depends. The pulse is not necessarily irregular, the quantity of fluid being commonly insufficient to affect the heart's action very seriously; while the constrictive influence of exudation-matter, and any dynamic perversions of muscular action, dependent on contiguous inflammation, are altogether wanting. Orthopnœa may occur; but if so, as far as I have seen, double hydrothorax exists to share in its production.

463. *Treatment.*—Diuretics and hydrogogues seem to have less effect on this dropsy than on others; such medicines will, however, of necessity, be tried, were it only for the removal of the usually concomitant dropsies. Cautious cupping, or dry-cupping over the heart, would be advisable, if the symptoms became urgent. A blister has sometimes appeared to me useful.

Paracentesis has been performed, with temporary relief; but, unless the primary disease be removed, the fluid will of course be reproduced. The suggestion of Laennec, that slightly irritant fluids should be injected into the pericardium, so as to excite an inflammation that might, by causing adhesion, prevent the recurrence of the disease, has acquired more claim to serious consideration, since the experiment of injecting an ioduretted solution in hydropericarditis has been successfully tried [401].

III.—PNEUMOPERICARDIUM.

464. Pneumopericardium is the term devised to signify the presence of gas in the pericardial sac,—no other abnormal product or appearance being discoverable. The state may conceivably be of *perforative* or *secretive* mechanism.

465. *Perforative.*—Now pneumopericardium must exist temporarily, be it for ever so few minutes, as the sole result of perforative

communication between the pericardial sac and any hollow viscus containing gas; but in this isolated state it has never been observed,—pericarditis having supervened before clinical examination has been made. A rare case is recorded by Flint.[*]

466. *Secretive.*—As to secretive or idiopathic pneumopericardium, I am not aware that its existence has ever been conclusively shown after death, much less that it has been diagnosticated during life. Laennec's expressed conviction that in almost all cases, when the heart's action is heard at a distance from the body, the cause of the phenomenon is a temporary development of gas in the pericardium, cannot at the present day be received.

IV.—PNEUMO-HYDROPERICARDIUM.

467. Laennec taught that effusion of air and serum into the pericardium may occur during the closing struggle in all diseases. He states he had sometimes announced its presence, on the evidence of increased resonance over the lower part of the sternum, and of præcordial fluctuation-sound caused by the heart's action and by deep inspiration.

There is reason to believe Laennec deceived himself as to the frequency with which gas and simple serum are to be found in the pericardium. For my own part I have met with few positive examples of the kind,—perhaps I have not been sufficiently watchful. Here is an instance in point occurring in a man, aged forty-seven, cut off by phthisis, which had largely excavated the left lung: the following conditions were noted,—

> "P.M., forty-six hours after death, June 24, 1858, weather dry and hot.—Pericardial sac considerably distended with gas, giving true tympanitic note on percussion; none of the gas forced away by percussion" [hence the inference that the pericardium was not perforated]; "a small opening being made in front of sac, this gradually collapses, no fluid escaping: upper edge of the sac reaches the level of the first rib; the point of the heart corresponds to the fifth interspace; five and a half ounces of blood-stained serosity in sac, without a particle of lymph; the gas having once escaped, no bubbling through the fluid lying in the back part of the sac follows; the fluid in the sac acid at first; same litmus plunged into it again shortly after removal of the fluid from the sac, becomes blue after a moment's exposure to the air."[†]

Now there were sundry evidences of putrefaction in different parts of the body; still, much more advanced cardiac decomposition is frequently met with unaccompanied with gas in the pericardium. Hence there must have been something special in the case.

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[*] Dis. of Heart, quoted by Beghie, *loc. cit.*

[†] Thunder, U. C. H., Males, vol. xv., p. 79; also Crocott, U. C. H., Males, vol. xviii., p. 113.

V.—PNEUMO-HYDROPERICARDITIS.

467. So far we have referred to the presence of air in the pericardium independently of previous pericarditis. Now gaseous fluid may also conceivably accumulate in an inflamed pericardial sac in three different ways. (*a*) Gas may appear as an actual formation by the irritated membrane; (*b*) it may result from decomposition of liquid effusion; or (*c*) be conveyed into the sac by perforation, traumatic or diseased, from a neighbouring hollow canal or viscus.

(*a*) Stokes briefly describes a case of pericarditis, in which for three days metallic gurgling and crepitating sounds were heard over the cardiac region in accompaniment with amphoric and cracked-metal note on percussion,—the stomach not being distended with air and the lungs and pleura unaffected. The heart's sounds were audible at a distance. It seems equally difficult to contest the dependence of these phenomena on the presence of air in the sac, and to explain such pneumatosis by acknowledged pathological laws. There was no *post-mortem* examination.

(*b*) The fluid of pericarditis, fœtid and decomposed, has been known to furnish gas during life. In a case of this sort, observed by Bricheteau, the heart's action was accompanied with a "sound like that of a water-wheel." The actual presence of gas in the sac was ascertained after death.

(*c*) In the singular case of traumatic communication between the œsophagus and pericardial sac, at some of the particulars of which I have already had occasion to glance [76], the following were the main physical signs I succeeded in substantiating. The percussion-note over the heart-region was purely tympanitic, not in the least tubular or amphoric,—a quality I take as demonstrating that the proportion of gas to fluid in the sac was largely in excess. No gurgling noise accompanied the cardiac action, nor were the heart's own sounds, or the existing friction-murmur, particularly loud. The really distinctive phenomenon consisted in the change of position of tympanitic and dull percussion-sound, within the area of the cardiac region, according as the posture of the patient was changed from one to the other side.*

W. Begbie reports a very remarkable case, wherein he succeeded in diagnosing pneumo-pericardium (with antecedent and sequential pericarditis) resulting from cancerous perforation through the œsophagus into the serous sac.†

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* Ramo Samee, U. C. Museum, No. 3859.

† Edin. Med. Journal, Oct. 1862.

Graves records a case of fistulous communication between a hepatic abscess and the stomach on the one hand and the pericardial sac on the other. Loud metallic ticking with each stroke of the heart, combined with friction-sounds and a noise like emphysematous crackling, were the signs of the pneumo-pericarditis following perforation. M'Dowel has observed a case of communication between a cavity in the left lung and the pericardium; metallic tinkling, amphoric buzzing, and splashing of fluid were caused by the action of the heart. (Stokes, op. cit. pp. 23, 25.)

§ VII.—ALTERATIONS OF NUTRITION.

I.—Atrophy.

468. Atrophy may be limited to some portion of the valvular apparatus, or to the muscular structure.

A.—VALVULAR ATROPHY.

469. (*a*) When the cordæ tendinæ of the mitral valve are very short and extremely thin, they are, probably, purely atrophous. The larger tongue of this valve is sometimes simply defective in size, without obvious puckering, or other evidence of past inflammation. In both cases, regurgitation may occur. Reticulation of the mitral valve is rare.

(*b*) The tricuspid valve is sometimes at once deficient in depth, its substance thin and papery, and its cords excessively delicate,—conditions apparently proving deficient nourishment, and the first of them certainly tending to promote the occurrence of regurgitation through the orifice.

(*c*) The sygmoid valves, both pulmonary and aortic, may be thin and papery; whence a sharp clicking state of the second sound, but no actual disturbance of the heart's action, ensues.*

VALVULAR RETICULATION.

470. The sygmoid valves are, besides, the tolerably frequent seats of so-called reticulation or cribriform perforation. The minute foramina, giving the sieve-like appearance, almost always lie close to the free edge of each valve, except at the spot

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* This change in the quality of the second sound furnishes no argument in favour of the theory ascribing that sound to valvular vibration. The change occurs simply because the fall of the blood-columns must emit a different kind of noise, when falling on comparatively thick and thin layers of resisting material.

corresponding to the corpus Arantii; they vary in size from that of a pin's head to a slit comprising the entire length from the corpus Arantii to the attached edge of the valve; they may be many in number, or but one; and vary in form—round, oval, slit-like. Sometimes the perforation is imperfect, the intra-serous connective tissue only being wanting, while the proper endocardial structure seems sound. All the cardiac textures may be otherwise thoroughly natural, even when this reticulation is carried to an extreme point.

Bizot* was the first to notice that the pulmonary and aortic valves presented this kind of defect with about equal frequency. He further ascertained that it is more frequent in males than females; and that, rare before the age of fifteen, it suddenly increases in frequency at and just after that age, and ceases to grow more common at and after the age of forty.

471. *Nature.*—Now, reticulation of the valves is here spoken of under the head of atrophy, to avoid multiplying divisions; it seems very doubtful whether the condition be truly, or at least simply, atrophous. It can scarcely be conceived to be a fatty form of local waste, because it implicates the two sides of the heart with such closely equal frequency. On the other hand can we accept as satisfactory, ingenious though it be, Bizot's theory, ascribing the perforations to rupture from extension by the rapid increase in the heart's dimensions, which sets in at the period of life when reticulation first becomes common?

472. *Symptoms.*—Interesting enough in an anatomical point of view, this sieve-like condition proves clinically unimportant. It produces no subjective annoyances of any kind.

473. *Physical Signs.*—On first thought it seems conceivable reticulation might cause regurgitant arterial, that is basic diastolic, murmur. But I have never known a murmur actually so produced; and if its occurrence were usual, the murmur signifying pulmonary regurgitation ought, instead of being one of the *mirabilia* of clinical practice, to be common,—seeing that reticulation is very closely as frequent in the pulmonary, as in the aortic, valves. Besides, there is an anatomical cause why, unless in extreme cases, reticulation should have no disturbing influence on the circulation; it affects those parts of the valves, close to their free lunated edges, that lie vertically surface to surface in the centre of the vessel, at the moment of its systole;—these particular portions of the valves have nothing directly to do, physiologically,

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* Mémoires de la Soc. Méd. d'Observation de Paris, t. i., 1837.

with the prevention of regurgitation. It is, perhaps, barely possible, however, as already suggested [115], that the peculiar basic sound, symbolised by the syllable *phvē . . tt*, might be thus engendered,—reflux of blood taking place at the *commencement* of the act of closure.

B.—MUSCULAR ATROPHY.

(I.) CONCENTRIC FORM.

473. The heart is said to be the subject of concentric atrophy, when the size and weight of the organ and the capacity of its cavities are alike diminished.

474. I have been led in but a single instance to imagine I had met with an *idiopathic* example of this affection during life. Here no diathetic disease of any kind existed; the symptoms and physical signs solely indicated smallness and feebleness on the part of the heart: but I want the *post-mortem* proof of the correctness of the diagnosis. Congenital smallness of the coronary arteries may conceivably act as a cause of such defective nutrition.

475. I have seen an atrophous state of the left ventricle (as others had done before me), produced by the close embrace of pericardial induration-matter [397-8]. But so far from such embrace habitually entailing atrophy, it seems rather to stimulate the nutrition-process in the organ. Accidental excess of pressure on the coronary arteries possibly explains atrophy, when it occurs. Atrophy, it is alleged, has sometimes been artificially produced by the means adapted for the treatment of hypertrophy,—a statement which, although emanating from Laennec, still requires corroboration.

476. In phthisis and in carcinoma, as originally shown by Louis and Bizot, the weight and bulk of the heart fall, as a rule, sensibly below the average. So true is this, that the sustainment of the heart's weight at the par of health, in persons slowly cut off by either of these diseases, may in my apprehension be looked upon as the equivalent of hypertrophy in people free from the one or the other [36].

The heart has occasionally been found in an atrophous condition in persons cut off by constitutional syphilis (*vide* SYPHILOMA).

Now the association of such atrophy with syphilis and especially cancer, as well as with phthisis, would almost suffice, taken alone, to disprove the theory credited to Brehmer, which reversing the order of relationship generally received, ascribes phthisis to smallness of the heart (Syd. Soc. Retrosp. 1869-70, p. 145).

477. *Symptoms.*—No subjective symptoms can be traced with positiveness to this condition of the heart. Palpitation has sometimes been observed, but in carcinoma is probably rather the result of attendant spanæmia than of the atrophy. It would appear that cardiac paræsthesiæ of different kinds may occur;—I have observed the coincidence, but cannot feel sure of the real connection of cause and effect. The pulse is small and wanting in vigour: other characters, appertaining to those diseases, affect it in cancer and phthisis.

478. *Physical signs.*—The heart's impulse is deficient in force, and materially limited in area both to the eye and hand. The extent of præcordial percussion-dulness, both superficial and deep-seated [70], ranges very materially below par. Thus in the apparently idiopathic case above referred to, percussion of medium force failed to elicit cardiac dulness over a space larger than from one half to two-thirds of that fairly to be looked for in an individual of the same stature possessed of a heart of normal dimensions. It is important to add, there was neither emphysema, nor any other condition capable of interfering with the heart's percussion, perceptibly present. Of the character of the cardiac sounds I know nothing of importance. In this case they were not appreciably abnormal.

(II.) ECCENTRIC FORM.

479. When the walls of the heart are greatly attenuated with or without subsidiary dilatation, loss of mass has occurred, and the state may fairly be termed one of eccentric atrophy: the fibre is deficient in firmness. This condition is singularly rare; and is rarer still in the left than the right ventricle. In the latter situation it probably intensifies some of the effects of the dilatation and tricuspid insufficiency, with which it is commonly associated; but, in point of fact, I have seldom seen atrophy of the kind.

(III.) LIMITED OR LOCAL FORM.

480. This form of malnutrition is far from uncommon. In this point of view it would call for full description; but on the other hand, in its anatomical conditions and functional effects, it is so closely associated with certain other changes of texture, that its history may be most advantageously given with the account of

these changes,—namely, fatty infiltration, fatty metamorphosis, and cirrhosis.

481. The heart's fibre is very certainly, to say the least, but little susceptible of that intrinsic form of decay, unattended with adventitious production of any kind, known in the voluntary muscles as "Cruveilhier's progressive muscular atrophy."* Still local waste of analogous character does, it is affirmed, sometimes appear to occur; but, singularly enough, not when the external muscles are affected. There is nothing known concerning this state clinically,—if, in truth, it has a real existence.

II.—HYPERTROPHY.

A.—VALVULAR HYPERTROPHY.

482. Thickening, more or less notable, with opacity, of both the mitral and aortic valves, is sometimes observed in connection with hypertrophy of the left ventricle, where no anatomical or clinical indications exist of bygone inflammation; and where, in all probability, the thickening results from excess of nutrition consequent on the extra-work entailed on the valves by the muscular hypertrophy.

483. This state, if affecting the sygmoid valves, impresses a dull, heavy, clanging character on the second sound: this is matter of observation, and is a perfectly intelligible effect on the recoil theory adopted in the first part of this work [96]. A similar anomalous condition of the first sound, where the mitral valve is notably thickened, probably arises from coexistent muscular hypertrophy.

I am not aware of ever having met with a case in which valvular thickening was carried to such a point as, unassisted, either to throw a difficulty in the way of the onward current sufficiently serious to generate obstructive murmur, or to interfere so effectually with the closure of the valves, as to cause regurgitant murmur. But it is well conceivable that, where such murmurs are produced by other causes, valvular thickening must intensify them.

B.—CARDIAC HYPERTROPHY.

484. In a certain share of cases of hyper-nutrition of the muscular substance of the heart, the affected walls and their

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* Roberts teaches the disease strictly confines itself to the muscles of the life of relation (On Wasting Palsy, p. 152). But how is the statement to be reconciled with the fact, that the diaphragm is certainly implicated in some cases?

enclosed cavities retain the relative proportions of nature,—as the former thicken, the latter *pari passu* widen: in such cases the hypertrophy is termed *simple*. Or while the muscular substance of, say, a ventricle increases in mass and density, the area of its cavity disproportionately enlarges; the hypertrophy is then styled *eccentric* or *dilated*. Or, lastly, while the muscular mass grows, it may encroach upon and actually lessen the capacity of the cavity it bounds: hypertrophy of this sort is known as *concentric* or *contracted*.

Hypertrophy may be *general*, that is, affect all the compartments of the heart; in this case it is always, as far as I have seen, of the dilated or eccentric species: or the extra-nutrition may be *limited* to one or two compartments; under these circumstances the species varies.

Whether alone, or in association with other portions of the organ, the left ventricle is by far the most frequent seat of hypertrophy; next comes the left auricle, closely followed in point of frequent implication by the right ventricle; while *longo intervallo* the right auricle completes the series.

The thickness of the wall of the left ventricle may increase from the normal amount of half-an-inch to two inches, according to some observers: I have not known it exceed one and a quarter inch exclusive of the columnæ carneæ. As a general rule, the basic part of the ventricle thickens most, the apex-region relatively least: in some rare cases of old standing aortic regurgitation, however, the precise converse is observed.

The columnæ carneæ, and especially the papillary muscles, thicken variously: if there be notable dilatation they are flattened, and may appear attenuated, though really extra-nourished.

The septum, enlarged similarly, encroaches on the cavity of the right ventricle, which becomes elongated, narrow, and concave, on the side next the prominent septum.

But not only may hypertrophy of a given compartment be or be not associated with contraction or dilatation of its own cavity, with one or other of the three species of hypertrophy in certain other divisions of the organ, but also with simple or attenuated dilatation of certain of those other divisions. The varieties of conceivable combinations of hypertrophy and attenuation, of dilatation and contraction, are hence obviously very numerous. Nature, however, actually produces only a limited few of the possible total number. Hope suggested an arrangement in order of frequency of those clinically known, to which I cannot help thinking exception may on more than one point be taken.

As far as my own observation goes, the most common original seat of hypertrophy is the left ventricle; a similar state, carried to much less amount, more or less rapidly, follows in the auricle, while dilatation advances in both. And clinically these morbid conditions may long remain confined to the left side; though before death, generally speaking, the right divisions have become implicated, so as to account for the fact, that general dilated hypertrophy holds the first place in frequency in Hope's table founded on the numerical results of *post-mortem* examination.

The physical conditions of an over-nourished heart vary of course with the capacity of its cavities. The superficial bulk of the organ does not exceed that of health in the concentric form, equals and exceeds it in the simple variety, more or less enormously outstrips it in the dilated species. In the latter it is that the general mass may be augmented to double, treble, even quadruple the normal amount: I have known the organ weigh forty ounces;* and entertain no doubt, on the evidence of physical signs, of having met with still greater increase of weight in some instances, where the opportunities for *post-mortem* examination did not present themselves,—as, for example, in the case referred to in the next paragraph but one.

In simple hypertrophy the special form of the heart is retained; while just in proportion as dilatation is superadded does the shape become unnatural,—spherical, square, or even broader than long. The apex under the latter circumstances is so rounded off, that it ceases to be distinguishable.

In regard of position in the chest, though the hypertrophous heart is habitually lowered, as an effect of mere weight, it occasionally becomes fixed at the normal level by pleuro-pericardial adhesions. The enlargement then affects the upper limits of the cardiac region; the organ reaches the second cartilages above, and the eighth rib below, stretching vertico-diagonally from nearly two inches to the right of the sternum to two and three-quarter inches outside the vertical line of the left nipple. In very rare instances of dilated hypertrophy, especially where the thorax is deficient in transverse width, the heart may *literally occupy the greater part of the left lateral regions of the chest and quoad percussion-results simulate pleural effusion.* †

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* Allen, U. C. H., Males, vol. xi. p. 346.

† This is an unexaggerated statement of the conditions in a case which once fell under my notice.—C. R. August, 1855.

The coronary arteries, one or both according to the seat of augmented growth, enlarge. Robert Lee states that the nerves and ganglia in the substance of the heart also increase in mass. Cloetta,* following in the inquiry, admits the enlarged size of the nerves, but professes himself in doubt whether the enlargement is due to the actual nervous substance or the fibrous tissue.

485. The walls of the aorta thicken, and the calibre of the vessel widens. The latter change is well shown at the orifice, which, if the right side of the heart be relatively ill-nourished, may very considerably exceed the pulmonary in width. These extra dimensions may certainly be found in the innominate and large vessels at the base of the neck also,—and in some instances the inferior thyroid arteries enlarge (*vide* CARDIO-THYROID EXOPHTHALMOS). It has even been affirmed that the entire arterial system widens; but hypertrophy is commonly a disease of middle and advanced life,—in other words of a period when, as to all seeming satisfactorily demonstrated by Bizot, the arteries normally increase in calibre. More probable is it that thickening of the walls of the systemic arterioles and capillaries follows,—but this has not been shown to occur, at least beyond the possibility of ready cavil, independently of diathetic disease (p. 290, *note*).

There is no sufficient evidence to prove that hypertrophy causes atheroma, calcification, or fissuring of the arteries; but the association occasionally observed of calcification of the pulmonary artery with dilated hypertrophy of the right ventricle (each of them so rare) seems to exhibit a relationship of causality.

486. The microscopical characters of the individual muscular fibre in hypertrophy do not deviate from those of health; and, as the primitive fasciculi do not increase in thickness, new ones are obviously formed. Production of non-striated fibre, or even of fibre more imperfectly striated than in health, appears not to occur. The direction of the fibres sometimes undergoes slight change, evidently from lateral pressure of new fasciculi irregularly produced; the change is insufficient to modify on any efficient scale the natural line of action of the fibres [50, *a*]. Firm hypertrophy in one part of the walls of a ventricle may be associated with softness and anæmic tint, or with fatty change, in another; an anatomical fact which frequently proves of clinical importance, by modifying the manner of impulse of the enlarged organ. Even in the firm variety, the hardness and tenacity of each

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* Virchow's Archiv. 1853.

individual fibre do not appear to undergo increase; close packing leads to simulation of augmented hardness.

487. *Causes.*—The causes of hypertrophy of the different compartments of the heart may be grouped in the following manner. In some instances the reality of the assumed influence is more or less doubtful,—a fact, signified in each instance by a note of interrogation.

I. *Causes originating in the system at large.*—Excessive nourishment, especially nitrogenised, combined with free use of stimulants, and sedentary habits; excessive exercise, as pedestrian, rowing (and that of trades straining the upper extremities in excess ?).

II. *Causes originating in the blood.*—Uræmia ? rheumatic hyperinosis ?

III. *Causes originating in the heart itself.*—(*a*) *Functional*: Excitement of the heart, as the habitual palpitation of prolonged anæmia, and of cardio-thyroid exophthalmos; irritation of chronic pericarditis [393—8]. (*b*) *Mechanical Obstruction:* (1) *Affecting left ventricle:* Aortic constriction, aortic regurgitation, mitral regurgitation, mitral constriction ?[*] mitral regurgitation and constriction combined. (2) *Affecting right ventricle:* Tricuspid regurgitation, pulmonary constriction, mitral regurgitation indirectly through engorgement of the lungs. (3) *Affecting left auricle:* Mitral regurgitation and constriction, aortic regurgitation and constriction in much less degree. (4) *Affecting right auricle:* Tricuspid regurgitation.

IV. *Causes originating in the great vessels.*—Pressure obstructing their interior; smallness or constriction of aorta; aneurism of aortic arch near the heart ? ? (Aneurism of Aorta [779, *h*]); diminished elasticity of coats of aorta or pulmonary artery, affecting severally left or right ventricle ?

V. *Causes originating in the lung-circulation.*—*Affecting right ventricle:* Chronic bronchitis; emphysema; contraction after pleurisy; dilatation of bronchi and cirrhosis of the lung; diminution of cavity of chest, by deformity or by pressure of abdominal tumours ? It is possible, too, that the left ventricle may eventually become hypertrophous from the strain thrown on it by the systemic, sequential to the pulmonary, obstruction.

VI. *Causes originating in the kidney.*—Persistent obstruction in its capillary circulation ? ?

VII. *Causes originating in systemic capillary obstruction.*—Prolonged obstruction of any given mechanism in some large portion of the systemic capillaries ? (*vide* V.)

The most easily intelligible of all these modes of production of the disease seems to be that depending on mechanical difficulty at some one of the orifices; but no direct ratio constantly holds between the amount of hypertrophy and of valvular obstruction,—showing that even under these circumstances there is a something beyond mechanical difficulty which contributes its quota of causation. And in cases of this form of mechanism the overgrowth of the heart is less to be looked at in the light of a disease, than as the healthy result of the natural effort to supply extra power to overcome extra difficulty: hypertrophy of this kind may be called *balancing*, *compensating*, or *supplementing*.

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[*] Mitral constriction may be hypothetically supposed to cause left ventricular hypertrophy, either through the effort of the ventricle to overcome the systemic capillary obstruction consequent on the pulmonary obstruction, immediately induced by the mitral constriction; or through the extra-effort made by the ventricle to propel the small quantity of blood supplied by the auricle, to make up, as it were, by force of propulsion, for smallness of supply.

Males are in a very considerable ratio, at least that of 2 : 1, more subject to the disease than females,—probably rather because they are more exposed to its exciting causes, than from any inherent sexual peculiarity. Very much on the same principle is to be explained the increased frequency of the disease with advancing years: well marked hypertrophy may, however, exist in earliest childhood, especially in connection with valvular defect.

488. *Symptomatology.*—The symptoms, signs, and effects of hypertrophy of the different compartments of the heart differ so materially, that, to avoid confusion, it will be advisable to consider the disease in each situation separately. Nor must it be forgotten that pure and simple examples of hypertrophy are rare; that valvular affections commonly exist to modify both the subjective, and especially the objective, effects of enlargement.

(I.) Hypertrophy of the General Substance of the Left Ventricle.

489. The student is to assume that, in the following description, the hypertrophy is either actually pure (this is, on the whole, very rare), or associated only with such amount of dilatation as shall be insufficient to throw its own special attributes into the shade. A well defined case is taken as the model.

490. *Physical Signs*—(*a*). Inspection discloses arching of the præcordial region (especially in long-standing cases, and in early youth), with widening, but without bulging, of the left interspaces, from the third to the seventh.

(*b*) The palpable impulse, increased in extent, especially to the left of the sternum, presents its maximum amount below and about the left nipple, and between this and the sternum; in character it is slow, heaving, and suggestive of pressure forwards steadily against an obstacle; in rhythm regular, unless there be some added morbid state; in force unequal. The amount of force may be sufficient to shake the head of the observer, the trunk of the patient, or the bed even on which he lies; such extreme power of action is rare, unless dilatation be combined with great hypertrophy. Of double (systolic and diastolic) impulse, I have already spoken [58, *a*]. The point of the apex-beat, carried downwards and outwards, may reach the lower edge of the seventh rib (rarely, however, without dilatation), at some distance outside a line let fall perpendicularly from the nipple. In eccentric hypertrophy the extent of visible impulse is much greater; the apex-point may be carried to the seventh space or eighth rib; the impulse may, without much difficulty, be felt in the back; its character is less heaving than in the pure disease, sharper, more knocking, or slapping, and the surface, over which it is perceptible to the hand, proportionally more extensive.

(*c*) The superficial and deep-seated dulnesses of the heart are both augmented in area, and, probably, in amount also,—the parietal resistance certainly seems sometimes very notably increased. In dilated hypertrophy, the dulness may reach from the second to the eighth rib,—proving marked enlargement in an upward as well as downward direction. Transversely cardiac dulness may stretch from an inch and a half or two inches to the right of the sternum to three inches and even upwards outside the vertical line of the nipple: and deficiency of pulmonary resonance may be detected in the back to the left of the spine. Nay, more, in those exceptional instances of huge bulk a moment since referred to, the percussion sound may be toneless and high-pitched all over the left lateral regions of the thorax, and vocal fremitus null, just as if pleural effusion existed.

The rudely triangular form, natural to the heart's *superficial* dulness (*vide* Diagram, p. 3), gives place to a dulness of somewhat square outline; and the deep-seated dulness is also more right-angled than in health;—this latter character, however, is not often to be satisfactorily ascertained.

In estimating the percussion-dulness really depending on a hypertrophous heart, the observer must bear in mind, that its apparent extent may be, on the one hand, increased by blood-engorgement of the right cavities, by aneurism of the aorta, by indurations in the lungs, pleura, or mediastina, by tumors of the œsophagus even, and by enlargements of the liver, which, by pushing the organ upwards and to the left, widen the area of dulness in those directions;—and, on the other hand, decreased by emphysema and bronchitic distension of the lung.

(*d*) The state of the cardiac sounds differs with the species of the hypertrophy. First, in the *simple* form the first sound is dull, muffled, prolonged, weakened in some cases almost to actual extinction, directly over the ventricle, the sensation reaching the observer's ear being rather one of impulsive motion than of sound;—under these circumstances a tolerably full systolic sound may, nevertheless, frequently be found at the base and at the ensiform cartilage; the extent of its transmission towards the apex is very limited. The second sound, lower pitched than natural, is fairly loud and of clanging quality. The post-systolic silence is shortened. During palpitation the first sound sometimes becomes comparatively full-toned. Secondly, in hypertrophy with dilatation, the sounds gain greatly in loudness and extent of transmission, especially if the valves be perfectly healthy and free even from hypertrophous thickening; and the tone of the first at the left apex is notably higher-pitched than natural. Reduplication of either sound, sometimes occurring, possesses no special character: it is not common,—especially when the inequality of strength in the two ventricles is considered.

Systolic blowing murmur, basic and audible at the second right cartilage, is sometimes heard in cases of pure hypertrophy; nor can it be positively ascribed in all instances to coexistent spanæmia. I have known such a murmur disappear, when the heart had become comparatively quiescent by treatment of a kind depressing rather than otherwise, and wholly non-ferruginous. Hence excess of force of propulsion of naturally constituted blood would seem capable of generating *direct* murmur (*vide* Dynamic Murmurs, p. 88). Again, hypertrophy may possibly, during the excitement of palpitation, induce mitral regurgitant murmur by disturbing the action of the papillary muscles,—especially as these muscles are often relatively nourished more, sometimes less, than the rest of the ventricle, sometimes even more or less extensively cirrhosed. Such murmur actually does, as a clinical fact, exist at one time and disappear at others.* And systolic basic murmur may also, very probably, be generated in cases of dilated hypertrophy, in consequence of the altered relationship of the aortic orifice to the cavity of the ventricle—altered both in point of size and of direction of the blood-current [106, *n*]. In dilated hypertrophy, knocking and rubbing additions to the first sound at the apex, either left or right, are not very uncommon. Possibly, too, the second sound may be, in seeming at least, intensified at the left apex by the abrupt recedence of the enlarged heart from the side during its diastole [98].

The main axis of the heart, when any notable dilatation is conjoined with the hypertrophy, becomes more horizontal than natural. It has been supposed the great vessels may consequently undergo more or less marked twisting at the base, and systolic murmur arise in consequence. I have never known murmur demonstrably thus caused; the effects of lateral detrusion of the heart in pleuritic effusion, while they show such an effect is not impossible, would lead us to expect that it must be very rare.*

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* Bousey, U. C. H., Males, October, 1850. Slight systolic murmur existed at the left apex in this case when the heart was much excited, disappearing wholly when its ordinary beat had been calmed down by treatment.

The respiration at the centre of the cardiac region, that is, the upper sterno-costal angle of the fourth left interspace, is feeble and distant,—but not so feeble nor so distant as it would be with an equal amount of percussion-dulness from fluid in the pericardium.

491. *Symptoms and state of the functions generally.*—The state of the functions may be described as follows, in cases of pure hypertrophy, or hypertrophy without any notable amount of dilatation,—the excess of nutrition being besides either wholly limited to the left compartments, or involving the right to a comparatively slight extent.

(*a*) The strength does not seriously suffer, unless the disease be carried to a great height; the power of walking and of ascending hilly ground is diminished, not from feebleness, but from the dyspnœa and oppression induced by the attempt. Patients generally sleep and recline with the head high.

(*b*) The colour of the integuments varies; if the hypertrophy be pure, the face is florid; if coupled with moderate dilatation, there may be slight purpleness and lividity; but marked purple discoloration does not occur, unless there be very considerable dilatation, valvular obstruction, or pulmonary disease. Hypertrophy of the left ventricle does not *per se* produce œdema of the ancles, much less general anasarca: even hypertrophy and dilatation, provided the latter be not in great excess, fails to induce this evidence of systemic vascular obstruction,—at least unless the disease has lasted for a long time, when all motive exists for supposing, or knowing, that blood-alteration has set in.

(*c*) The muscles are well-nourished and of good colour.

(*d*) There is not any form of dyspeptic derangement particularly assignable to hypertrophy; the disease may exist for years without materially affecting the primary digestive powers, provided moderate exercise be taken. Constipation acts as a source of habitual annoyance.

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* Diseases of the Lungs, 4th edit., p. 259.

(*e*) There is more or less dyspnœa, either constant or occasional,—in the latter case induced by the most trifling effort: the pulse-respiration ratio may be perverted in consequence; paroxysms of true cardiac dyspnœa have not occurred under my observation, unless there were much dilatation, valvular obstruction, or pulmonary disease. Dry cough annoys some patients; I have not observed œdema of the lungs, nor hæmoptysis.

(*f*) The radial pulse, in no wise peculiar in regard of frequency, and perfectly regular in rhythm, is full, strong, firm, tense, resisting, and prolonged, without jerk or thrill, in the pure disease; if dilatation be superadded, it retains its fulness, but loses in some measure strength and resisting power. It is said not to be increased in frequency, as in health, by change from recumbency to the sitting and standing postures. The sphygmogram in pure hypertrophy is remarkable for the height and verticalness of the line of ascent (*vide* APPENDIX). The action of the carotids is visible; and in aged persons the pulsations of the smaller superficial arteries may be generally distinguished by the eye [141, *b*]. Præcordial pain, rare in simple hypertrophy, is not uncommon in the dilated variety, ranging in severity from a slight aching sensation to the severe suffering of pseudo-angina. Paroxysms of such pain may be accompanied with, and probably depend sometimes on, congestion of the lungs and loading of the right cavities of the heart with blood; in some instances they are distinctly traceable to intercostal neuralgia. They rarely attain any notable amount of severity, unless valvular disease coexist.* The common action of a hypertrophous left ventricle would be palpitation, if not in frequency, in force, to a healthy person; under excitement, or often without apparent cause, a violent fit of throbbing action comes on,—regular, however, or almost so, in rhythm, producing forcible pulsation in the neck and head, with tinnitus aurium.

(*g*) No change, that I am aware of, occurs in the lymphatic vessels or glands.

(*h*) I have not known hypertrophy produce albuminous impregnation of the urine, nor indeed any distinctive condition of the fluid,—not even notable modification in the quantity, as compared with the amount of drink. The dynamic agencies, which may by possibility affect the renal circulation in cases of pure or dilated hypertrophy, are doubtless on the whole conflicting enough; yet it seems to me that if the doctrines, taught of late years concerning the influence of excentric pressure of the blood in the capillaries of the Malpighian tufts on the amount of water excreted, were perfectly sound, the mean daily discharge of urine ought to range more or less steadily and markedly above par.

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* Allingham, U. C. H., Females, vol. xvii., p. 53. But even with this associated condition any near approach to genuine angina is very rare in cases of decided hypertrophy [341, 346].

(*i*) It has been said the sexual propensity undergoes increase,—a statement I can neither affirm nor deny,—but one which seems to have been put forward on theoretical grounds.

(*k*) Cephalalgia, dull, aching, or throbbing, is of more frequent occurrence than in healthy persons, but by no means a constant symptom; sensations of rushing of blood to the head are common, especially on stooping, and, indeed, on sudden movement of any kind. The intellect is habitually unaffected, as regards any symptomatic state clinically significant; no proof exists of its being brightened; nor, on the other hand, unless towards the close of life, have I found pure hypertrophy render individuals incapable of ordinary mental exertion. A disposition to drowsiness, especially after meals, occasionally, but not by any means with the frequency that might be anticipated, proves a troublesome symptom.

(*l*) Reflex muscular phenomena, at the moment the patient drops off to sleep, sudden starting of the legs, for example, are not very uncommon; they may possibly be traceable to active congestion of the spinal chord.

(*m*) The eyes of some patients are bright, full, prominent, prone to injection (*vide* (*n*)); and by such persons visual illusions, luminous vision, and muscæ volitantes are frequently complained of. I am not aware that cataract or other textual change in the eye has been found of unwonted frequency in pure hypertrophy. Epistaxis seems to be more usual than in individuals of equal age free from hypertrophy.

(*n*) The causal relationship of purely hypertrophous heart and enlargement of the thyroid gland and inferior thyroid arteries with prominence of the eyeballs is considered elsewhere (*vide* CARDIO-THYROID EXOPHTHALMOS).

492. *Secondary morbid changes.*—Certain textural changes have been held to follow as a necessary consequence of the influence, long sustained, of a left ventricle over-nourished, and hence, by inference, powerful to excess in its contractions.

493 (*a*). Hypertrophy of the left ventricle has thus been said to entail *increase in weight and substance of the organs generally.*

Plausible enough though the proposition be, and supported though it be to a certain extent by some statistical returns of Clendinning, it cannot be looked upon as an established truth. I have seen rare cases in which the weight of several of the organs actually fell below par. But pure hypertrophy of the heart is difficult to meet with; and if fatty atrophy to any serious measure deteriorate the fibre of the generally thickened ventricle, the conditions of the problem are plainly altered.

(*b*) The great frequency with which morbid changes in the heart and the anatomical conditions of the diathetic affection known as "Bright's disease" are associated, is matter of universal recognition. But opinions are far from being unanimous concerning the relationship of the cardiac and renal states in point of sequence, yet more in point of causation.

Setting aside for the moment those much more common cases where some form of valvular disease coexists with the muscular extra-growth, and confining myself to instances of pure hypertrophy, dilated or not, but unattended by disease of the orifices sufficient to disturb the circulation notably, I find some few instances in my hospital-books where from the clinical course of events, as well as from the *post mortem* appearances, the renal changes must have preceded the cardiac in order of development,—none where the cardiac distinctly led the way,—some where the sequence could not be determined, and where probably the association was accidental: I take it then, we may admit that the renal disease is not caused by the cardiac.

Is the cardiac *per contra* caused by the renal? Bright, who first noticed the relationship of coexistence, seems to have supposed the struggle against the capillary obstruction in the kidneys sufficient to account for hypertrophy and dilatation of the left heart. I cannot accept this mechanical doctrine. My cases show me no steady proportion holds between the amount of capillary obstruction in the kidney and the cardiac hypertrophy.

Believing, as I have long done, on what appear logical grounds, that "Bright's disease" is primarily a diathetic blood-affection,* entailing, like all affections of the diathetic group, secondary alterations in the blood also, I look on the cardiac hypertrophy, occurring in its course, as: first, an additional local expression of the main diathesis; secondly, as a result of the specific irritation of the blood, primarily and secondarily altered in composition, on the endocardial surface; and, thirdly, as an effect of extra force expended by the left heart in overcoming a tendency to stagnation in the entire rete of systemic capillary vessels—itself caused by the altered qualities of the circulating blood and impaired elasticity of the vascular walls.[*]

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* "Bright's disease essentially and primarily a Blood-disease."—Clin. Lect., Lancet, July, 1849.

There may besides be some connection, through the fatty diathesis, between pseudo-hypertrophy or fatty enlargement of the heart and fatty alteration of the kidney; but I have seen nothing demonstrative of any such connection.

(*c*) Hypertrophy of the heart, and especially of the left ventricle, has long been supposed to play a very important part in producing certain *acute and chronic textural changes in the brain.* The following passage, now many years old, still seems to me to place the actually attained knowledge of the matter in the fairest point of view. In order to avoid the necessity of recurrence to the subject some other conditions of the heart are at the same time considered:—

"Nothing can appear more plausible than to suppose undue force of propulsion of the blood from the heart shall act as a cause of apoplexy, whether this be simply congestive (ictus sanguinis), hæmorrhagic, or dependent on red softening. Now, over-forcible propulsion of the blood through the systemic arteries is mainly the result of too energetic contraction of the left ventricle. Hence, hypertrophy of the left ventricle has been, by a process of *à priori* reasoning, set down as a cause of apoplexy in general by some persons; by others, of cerebral hæmorrhage, in particular; by yet others, of red softening. Again, the doctrine referring white or colourless softening of the brain to deficient nutrition, occurring independently of inflammation, has had many followers. Persons taking this view of its nature are prepared to recognize, in any agency interfering with the distribution of a just amount of blood to the brain, an efficient cause of the disease. Now, constrictive disease of the aortic orifice, and regurgitant disease of the mitral orifice, must have this effect on the cerebral circulation, and so, it is inferred, must act as causes of cerebral softening.

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[*] George Johnson has conclusively shown that the walls of the capillaries and arterioles, not only of the kidney, but of various parts and tissues, undergo thickening in the chronic forms of "Bright's disease." But whether this thickening really depend, as maintained by its discoverer, on *hypertrophy* of the muscular tissue of the affected vessels,—or on "hyalin-fibroid" formation in the walls of the minute arteries, and "hyalin-granular" change in the corresponding capillaries, often associated with "*atrophy* of the muscular coat," as recently asserted by Gull and Sutton, remains to be determined.

"It has been felt, I may almost say, on all sides, that these views require to be tested by facts; and the facts adduced, as far as they have reached me, may be summarily set forth as follows. First (*a*), in regard of 'apoplexy,' and 'cerebral hæmorrhage,' we find writers giving the subjoined numerical results of their observations concerning connection between those çerebral affections and heart-diseases.

	Deaths from Cerebral Hæmorrhage.	Heart Hypertrophous.
Rochoux	42	3
Andral	17	9
D. Fardel	28	8
	Apoplexy with Hemiplegia.	Heart-disease.
Clendinning	28	15
Hope	39	27
Burrows	34	23
	188	85

"Calculating from the sums of these somewhat contradictory results, there seems presumptive evidence that the heart will be diseased in about 45·2 per 100 of cases of apoplectic seizure with sudden hemiplegia. But the precise ages of the persons supplying these figures is unknown. That it was advanced in the great majority of cases may be admitted from the known laws of cerebral hæmorrhage; that it was advanced in Fardel's patients is certain, for they were all observed at the Saltpêtrière, where the rather patriarchal age of sixty years forms a condition of admission; the mean age in Andral's cases I calculate to have been 56·2 years. Now what proof have we that the number of hypertrophous hearts in the victims of cerebral hæmorrhage, just counted and percented, is greater than it would prove in a like number of aged people, cut off by all diseases, indiscriminately, affecting other organs than the brain? Absolutely none. So far as evidence goes, this seems, on the contrary, to show that the inference of close nexus of cause and effect, in cases where the cardiac and cerebral diseases were found, is illogical. Thus, Fardel carefully examined the bodies of sixty aged persons (at the same institution, the Saltpêtrière), cut off by other than cerebral causes, and found the heart sound in forty-five, hypertrophous in fifteen, of the number. In other words, we may expect that 25·0 per cent. of a mass of aged persons cut off by all diseases indiscriminately (except those of the brain), will have a hypertrophous heart. Now, this proportion of 25·0 per cent. is only twenty per cent. less than that of diseased hearts, furnished by the above series of apoplectic persons.

"I have separated the results of the six authors quoted into two sub-series. The separation is an important one enough. The first sub-series refers especially to 'cerebral hæmorrhage' in connection with 'hypertrophy;' in the second, the association is between 'apoplexy' and 'heart-disease,' no precise affirmation being made, or intended, as to the condition productive of apoplexy, or as to the nature of the heart-affection. It is a striking fact that the first sub-series gives a proportion of hypertrophous hearts of only twenty-three per cent.—actually two per cent. *less* than in aged persons wholly free from cerebral affection.

"Let us next examine the question in the converse point of view, and see in what proportion of persons having hypertrophy, or other disease of the heart, as their main affection, cerebral apoplexy supervenes. Here the following numbers are available for our purposes:—

	Disease of Heart.	Cerebral Apoplexy.
Ravier	10	1
Louis (Oral Lectures) . .	45	0
Blakiston	155	14
	210	15

"So that 7½ per cent. only of persons labouring under diseased heart, for a greater or less number of years, became the victims of cerebral apoplexy.

"Secondly (*b*), in regard of cerebral softening, the following figures may be adduced:—

	Brain softened.	Heart hypertrophied, or hypertrophied and dilated.	Aortic constriction.	Mitral regurgitation.
Andral . .	33	9	3*	—
D. Fardel . .	41	8	—	2
Rostan . .	18	12	—	—

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* One of these counts under the head of hypertrophy also.

"From these figures it would follow that 35·9 per cent. of persons having a softened brain had hypertrophy, or hypertrophy and dilatation of the heart,—scarcely more than Fardel's average for aged persons dying of all diseases indiscriminately. In the series just given, Rostan's figures all refer to old persons, and those of Fardel to persons aged upwards of fifty. The latter observer affirms that he always found the heart healthy in patients cut off by softening before the age of fifty. The mean ages in Andral's cases of softening may be calculated as follow:—Where the heart was sound, 49·9 years; where there was hypertrophy, or hypertrophy and dilatation combined, 47·7 years; and where there was constrictive disease of the aortic orifice, 63·7.*

"From this rapid survey of the subject of the connection of cardiac and cerebral diseases, what inference can fairly be drawn? None other, I think, than that positive asseverations of the power of hypertrophy to generate brain-affections, *as a demonstrated fact*, had best be avoided. On the other hand, I believe that it would be just as unsound to deny totally the existence of any such power on the faith of the numerical comparisons just instituted. These are the only comparisons of the sort obtainable at the present hour, but let us not shut our eyes to their serious imperfections. These imperfections are of different kinds. In the first place, some writers are so deeply prejudiced on the question at issue, that their facts cannot be received otherwise than with some quantum of distrust. Read the pages of Andral, and observe how determined he is to find the nexus of an hypertrophied left ventricle, and cerebral hæmorrhage; read those of Rochoux, and note the eager partisanship with which he strives to disprove it. Do this, and I feel satisfied you will, with me, see the wisdom of receiving *cum grano salis* their numerical conclusions. In the second place, some writers put forward cases where no *post mortem* examination took place; these are, under the circumstances, unfit to form elements in the discussion. Thirdly, who can for a moment suppose it to be at all likely, that all varieties of heart-disease shall have an equal tendency to produce cerebral congestion? That, for example, an hypertrophous left ventricle shall do the same violence to the minute cerebral vessels, if it play through a constricted aortic orifice, as if it play through a perfectly free one. Who can suppose that where the radial pulse is small, feeble, unequal, tremulous from highly developed insufficiency of the mitral valve, the blood shall be propelled with excess of force into the small arteries of the brain? Far from this, the contrary appears so fair an hypothesis, that Law regards such insufficiency as an efficient cause of gangrene-like softening of the brain [608, *b*]. The whole clinical history of mitral regurgitant disease points to pulmonary and not to systemic congestion. And *per contra*, the ascertained effects of tricuspid regurgitation give an air of probability to the view that this variety of cardiac imperfection may in reality form the true link between congestive affection of the brain and the heart. Whether this be the fact or not, time will show. Meanwhile, I think there can be no question that it is deeply unsound to club together cases of such opposite functional tendencies as those we have been noticing, and regard them as a single mass producing one single definite effect on the brain. Fourthly, an objection to some of the cases figuring in the returns given lies in the fact, that the respective dates of the cerebral and cardiac diseases have not been clearly made out. And again, fifthly, the subject would require revision, were it only for the change recently effected in our knowledge of the morbid anatomy of enlarged hearts. All enlarged hearts were formerly set down as enlarged by muscular hypertrophy; we now know that the available muscular substance may be less than natural in such hearts, encroached upon and impoverished as it is by accumulating fat. And let us not appeal here to clinical experience to set aside the inference deducible from *post mortem* investigation. True, the action, felt in the præcordial region, of a fat-infiltrated heart may be agitated and forcible, but violence of *impulsion* does not signify power of *propulsion*." *

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* Jordan draws attention to the occurrence of cataract in connection with disease of the mitral and aortic orifices, and with dilatation and fatty disorganization of the heart itself.—Brit. and For. Med. Chir. Review, vol. xix., p. 495.

A manifest connection exists between cardiac disease and cerebral embolism, leading to local death of brain-substance, paralysis of various kinds, and aphasia; but this connection belongs to chronic endocardial changes, to valvular disease, and to intra-cardiac thrombosis, not to hypertrophy.

494. *Prognosis.*—Simple hypertrophy, of medium amount, may be rendered a very endurable affection by means of regulated diet, moderation in exercise, and general attention to hygienic rules. If the disease be carried to a high point, if it be of considerable amount, and the patient, instead of living according to rule, be forced to work laboriously, and live irregularly, the probable issue is death, though secondary affections, complications and functional

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* Clinical Lectures, Lancet, vol. i., 1849.

derangements, all of which, though originally unconnected with the heart-disease, are rendered more serious by its existence. But pure hypertrophy alone is rarely, and I do not think ever rapidly, the direct cause of death: I cannot call to mind any case, where I have actually known it, and it alone, positively fatal. Hope, it is true, affirms he has known hypertrophy destroy life in some instances within a year of its commencement (Op. cit., p. 278); still he cites no cases, and appears from the context to have in his mind's eye examples of dilated hypertrophy variously and seriously complicated. Latham is of opinion, "the heart, *by the simple vehemence of its action*, has the power to kill,"—through cephalalgia, insomnia, delirium, mania, convulsion, and nervous exhaustion.* But by the phrase, "simple vehemence of action," we are not to understand, however this may appear warranted by its terms, such action as an unaided hypertrophous heart in its highest degree can engender. For Latham gives no proof that the organ possesses any such lethal power, and the only positive case referred to in illustration of the above opinion, is one of hypertrophy and dilatation of the left ventricle following endo-pericarditis and bygone dropsies;—where, too, as no *post mortem* examination took place, there can be no absolute certainty the encephalon was free from disease. Louis calculates the mean duration of hypertrophy of the left ventricle at twenty-eight months, and refers to a certain number of instances in which life was destroyed in from three to seven months. But the evidence is incomplete as to the purity of the hypertrophy; and it may be affirmed that the estimate of this accomplished observer falls below the mean standard observable in Great Britain.

The more dilatation predominates over hypertrophy, the more serious becomes the prognosis,—until at last the chances of existence and the quality and amount of suffering become very closely the same as in cases of unmixed, or almost unmixed, dilatation.

The influence of fatty metamorphosis of the fibre of a heart, previously purely hypertrophous, varies with the degree of each change. If the hypertrophy has been on a very extensive scale, bulk and action becoming dangerous in themselves, it is reasonable to suppose the weakening influence of fatty change would, up to a certain point, prove beneficial,—and in the course of years it has occurred to me to meet with an occasional example of such favourable influence during life, and to have the opportunity of proving after death the probable justness of the view now tendered. If, on the other hand, extensive death of true fibre occur through fatty metamorphosis, or even if encroaching subpericardial fat waste widely the sarcous structure by encroaching pressure, a lesser is simply exchanged for a greater evil [558].

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* Diseases of the Heart, vol. ii. p. 338.

495. *Treatment.*—With what hopes may the treatment of pure, or somewhat dilated, hypertrophy of the left ventricle be undertaken? Is the disease curable? Can the nourishment of the heart be not only controlled by artificial means, but reduced below the standard of health? Latham, Taylor, and Blakiston emphatically deny that art is possessed of any such power; Laennec and Hope maintain that the feat is easy of accomplishment. But Hope repudiates the plan recommended by Laennec, and the simple common-sense system lauded by himself fails utterly in effecting the textural changes he ascribes to it.

For my own part, I have never known the *cure* of indubitable hypertrophy proved by physical signs, or by *post mortem* examination, and hold it unwise to promise any such result from *treatment.* Even the physical signs may, unless he be very cautious, betray the observer into error in regard of this matter. Thus, not only the impulse may be reduced in force and extent, and the character of the first sound changed, but the area of percussion-dulness lessened, by disgorgement of the right cavities, and yet the heart's actual mass remain precisely as before. All this may sometimes be done, by treatment, in a few days; sometimes not in months,—but whether achieved quickly, or slowly, it gives no evidence that hypertrophy has been reduced.* On the other hand, it is not difficult to remove or greatly mitigate many of the symptoms of simple hypertrophy in the majority of cases, and render life not merely tolerable, but comfortable.

(*a*) The theoretical indication is very obviously to tranquillize the heart by diminishing the quantity, without deteriorating the quality, at least materially, of the circulating fluid. For this purpose, occasional bleedings from the arm, to the extent of four, six, or eight ounces at a time, at intervals of from two to six weeks, are recommended by Hope, in conjunction with a diet mainly consisting of farinacea and vegetables. But even loss of blood to the extent here intended, is more than can be borne by the majority of persons, without sequential impoverishment of the fluid; and the least amount of anæmia deeply aggravates the dangers of hypertrophy. Besides, it has appeared to me that over-action of the heart is quite as effectually and as lastingly controlled by very moderate cupping or leeching over the præcordial region, as by the abstraction of a comparatively large quantity of blood from the arm. Four or five leeches, even, will sometimes calm the excitement of a powerful left ventricle, in a state even of somewhat dilated hypertrophy,—and this in a well-grown adult. And, in a word, I never recommend venesection in cases of this class.

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* A youth, aged seventeen, came under my notice in 1862, with very distressing palpitation caused by competitive race-running at Rugby: the impulse proved to be somewhat heaving and the transverse percussion dulness above par, *especially to the left.* Eight years later I saw this patient again (Nov. 1870): he then suffered from grave paroxysmal cardiac seizures, but (the essential matter from our present point of view) "the heart's impulse was very feeble; and the area of deep-seated præcordial dulness *actually below par.*" Had hypertrophy spontaneously disappeared in this case,—or was its supposed existence at the first an error?

(*b*) There is no known drug possessed of the faculty of diminishing the bulk of the heart. Iodide of potassium, pushed even to the production of iodism, fails to exhibit any such power. And though I have an impression derived from tolerably careful watching of some few cases, that this agent may control the disposition to yet further increase, I should, I confess, be puzzled to produce demonstration that such impression is solidly founded.

(*c*) In point of fact, general principles seem the safest guides in the attempt to put a term to increasing bulk of the heart. As rest will waste the muscles of an arm or of a leg, so by analogy must rest, such as can be secured, weaken the nutritive activity of the heart. Quietude physical, emotional, and intellectual, is the very first of curative agents for an enlarged heart.

(*d*) To aid in tranquillizing the organ direct cardiac and nervine sedatives, aconite, veratrum viride, hydrocyanic acid, acetate of lead, and belladonna (the latter both internally and in the form of plaster to the surface) may be employed, with occasional intermissions, during the entire treatment of the case. Of all medicines of this class aconite, however, seems the best; the alcoholic extract of the root may be given in doses of one-eighth of a grain with perfect safety, provided its effects be watched from day to day; no drug, that I know of, possesses more fully the power of relieving painful sensations and disquietude about the heart [305, *a*]. The propriety of administering digitalis has already by inference been considered (p. 173).

Within the last few years I have employed the bromides of potassium and ammonium in cases of the present class—especially where dynamic excitation coexists in excess with the statical defect; their administration, as it has seemed, has been followed occasionally by very sensible reduction of the excitability of the organ, and *pro tanto* by prevention of further increase in its bulk.

(*e*) Purgative medicines, and I believe the saline and aloetic the most appropriate of the class, aid the good effects of rest and small local bleedings. Diuretics are useful, quite independently of the existence of dropsy.

(*f*) Unless the patient be very highly plethoric, animal food in moderation should be allowed,—under all circumstances, indeed, fish may be permitted. Alcoholic fluids, of all kinds, must be avoided; and liquid taken, as a rule, in but small quantity. Fatty food seems theoretically advisable.

(*g*) Walking exercise on a very limited scale is permissible; passive open air exercise more positively advisable.

(*h*) In treating a case of this kind, the patience of the physician must never fail him: it may require months, nay, years, to produce a favourable effect on the disease,—and want of steadiness of purpose and conviction may, in a few days, undo the good accomplished by the efforts of previous weeks. There is one caution to be given to the young practitioner,—that he never push any form of treatment to the extent of producing anæmia; the superaddition of anæmia to hypertrophy is that which gives a really ominous character to the latter.

(*i*) Whatever might be the forced *theoretical* argument in favour of attempting to relieve over-action by inducing fatty disintegration of some of the extra-muscular substance [494], the question need not delay us, as *practically* we are without the means of attracting such mode of alteration to the heart.

(II.)—CONCENTRIC HYPERTROPHY OF THE LEFT VENTRICLE.

496. The discovery that the contraction of the ventricle occurring *in articulo mortis*, increased by cadaveric rigidity,* might give to a heart simply hypertrophous the appearance of one concentrically hypertrophous, led, for a time, to the almost complete rejection of concentric hypertrophy as a possible state. But that its occasional existence is matter of fact, would be satisfactorily proved, were this even a solitary example, by a

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* When the spasm occurring *ante mortem* produces the semblance of concentric hypertrophy, it will be found that merely cutting into the tissue of the ventricle causes relaxation; it is unnecessary to have recourse to stretching or maceration. Case of Collins, U. C. H., Males, vol. vi., p. 1, Nov. 1850; death from dysentery.

preparation now before me.[*] Here, enclosed by walls equalling very nearly an inch in thickness, lies a left ventricular cavity, which would scarcely give lodgment to a flattened hazel-nut. There appears, besides, in this instance to be very notable relative excess in the thickening of the papillary muscles.

497. The *symptoms* and *signs* are those of simple hypertrophy. Theoretically the disturbance of the circulation will be greater, but clinical illustrations of the point are wanting. It has not occurred to me to meet with a pure case of the kind during the life of the sufferer.

(III.)—HYPERTROPHY OF THE RIGHT VENTRICLE.

498. Hypertrophy of this compartment, comparatively rare in all forms, is practically unknown except in association with a more or less dilated cavity.

The walls undergo quite as notable thickening, relatively speaking, as those of the left ventricle. In the adult the muscular substance may measure one, or even one inch and a-third in thickness; in a female child, aged ten years, I found it reach a quarter of an inch without, and seven sixteenths of an inch with, the columnæ carneæ.[†]

499. *Physical signs.*—Dilated hypertrophy of the right ventricle causes notable arching of the lower part of the sternum, with greater or less eversion of the ensiform cartilage, and fulness of the epigastrium. The left costal cartilages may be more bulged than the right,—a circumstance accordant with the fact, that though the impulse plays very forcibly against the sternum and ensiform cartilage, it inclines, in cases of highly marked right hypertrophy, to direct itself, in consequence of slight displacement of the entire organ, more against the edge of the left, than of the right mammary region. Hence it is, that the form of the præcordial region and the site of impulse might betray the observer into the notion that the left, and not the right, ventricle was the seat of hypertrophy. The percussion-dulness may extend considerably beyond the right edge of the sternum; in the child just referred to, it reached an inch outside that edge; at the same time the dulness may be carried unduly to the left also. The distended and enlarged right auricle contributes a large share of the percussion-dulness to the right of the sternum.

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[*] Univ. Coll. Museum, No. 2140. The reality of concentric hypertrophy has further been proved by Déchambre (Gaz. Méd. de Paris, 1844.)

[†] Kernis, U. C. H., Females, vol. ii. p. 237.

Dilated hypertrophy of the right ventricle widens the tricuspid orifice in the majority of cases; unless the valve grow in proportion to the widening of the orifice, a regurgitant murmur, inasmuch as the current is strong, should, theoretically, occur [110]. Yet, such murmur has certainly not been by any means constantly heard, in cases where, *post mortem*, the valve appears physically incompetent. Can this be explained by some constrictive action at the orifice during life which prevents regurgitation? The first sound is duller than natural at the ensiform cartilage; the second, as a rule, fuller, stronger, and more accentuated,—intensification of this sound at the pulmonary cartilage may, or may not (this is less common), be present [109]. Visible jugular pulsation sometimes exists, without the tricuspid valve being demonstrably imcompetent.

500. *Symptoms.*—Marked hypertrophy of the right, is so rare without hypertrophy of the left, ventricle, or some form of valvular disease, that it is difficult to give a really clinical transcript of its symptoms. Lividity of the face, and subcutaneous œdema about the face and neck, sometimes exist. Theoretically the lungs must suffer,—and hence dyspnœa, engorgement and œdema of the pulmonary parenchyma, bronchitis, pneumonia, tubercles, pulmonary apoplexy, and hæmoptysis, have all been set down by various speculative writers, as dependencies of the affection. Now, of all these alleged symptoms, dyspnœa is the solitary one that seems to have thoroughly made out its claim to be so entitled. I believe, however, that engorgement and œdema of the tissue of the lung is not uncommon. Pneumonia, pulmonary apoplexy, and hæmoptysis, I have certainly seen in cases of right ventricular hypertrophy; but in these instances, mitral regurgitation existed also. The notion that right hypertrophy tends to tuberculise the lungs, is not supported by anything I have observed.

The radial pulse, from its being free from all peculiarity of character, affords valuable aid in the diagnosis: its sphygmogram will corroborate the indications perceived by the finger. Natural in force, while the cardiac action is strong, the state of the systemic pulses argues against the existence of hypertrophy of the left ventricle, to which some of the local physical signs might otherwise point. The pulmonary artery, of course, bears the force of the hypertrophous ventricle; a fact explanatory of the frequency with which, under these circumstances, its coats become atheromatous and calcified.

The difficulty of circulation through the vena cava and jugulars tends to congest the brain venously.

501. *Duration.*—According to the results of Louis, hypertrophy of the right ventricle runs a mean course of six years and a half; this very remarkable prolongation of life, as compared with the experience of the same observer in regard of the left ventricle, is not easy of comprehension [494].

(IV.)—HYPERTROPHY OF THE LEFT AURICLE.

502. Hypertrophy of the left auricle often accompanies, in variable degrees, constrictive and regurgitant disease of the mitral orifice; but it has not fallen under my notice as an isolated state.

503. *Symptoms.*—The symptoms, whatever they happen to be, of these diseased conditions of orifice, are intensified by the overgrowth of the auricle; but there are not any disturbances known, which its hypertrophy can be said specially to produce.

504. *Physical signs.*—Abnormal deficiency of resonance to the left of the sternum about the third and second interspaces, and *impulse immediately preceding that of the ventricular systole*, are, as I have found, ascertainable physical signs.* It is possible too that knocking parietal sound may occur.

(V.)—HYPERTROPHY OF THE RIGHT AURICLE.

505. I have never seen pure hypertrophy of the right auricle; but hypertrophous dilatation, with chronic thickening of the endocardium, is often found, carried to a great extent, in cases of dilatation of the tricuspid orifice.

506. *Physical signs.*—Permanent extension of dulness at the upper right angle of the heart's area will accompany this state, and *impulse of præsystolic time* will, as I have had occasion to prove, be associated with the venous and other signs of tricuspid regurgitation.†

507. *Symptoms.*—As in the case of the fellow-compartment on the opposite side, there are no specially distinctive symptoms.

III.—CARDIAC DROPSY AND SEROUS FLUX.

508. Effusion of the serum of the blood into certain parenchymata and membranous sacs is one of the most important

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* F. Smith, U. C. H., Males, vol. v., p. 296, Oct. 1850.

† Mann, U. C. H., Females, vol. ii.

symptoms connected with various affections of the heart; serous flux, though of minor importance, calls for attentive study. And as the precise relationship of both to individual cardiac diseases must still be considered *sub judice*, the subject may, with propriety, be examined in a separate section.

A.—CARDIAC DROPSY.

509. Cardiac dropsies are preceded immediately by venous congestion, itself produced by conditions to be by-and-by inquired into. The fluid composing the effusion, of low specific gravity, contains but a small proportion of albumen, does not coagulate spontaneously, is free from urea, and, as far as known, any other excrementitious principle, and never forms a blastema apt for evolution.

510. *Site.*—Dropsy, sequential to heart-disease, occurs in the following situations, enumerated in the order of frequency with which they severally suffer: the subcutaneous cellular tissue; the pulmonary parenchyma; the peritonæal and pleural sacs; the pericardium; the cerebral and spinal arachnoid, and sub-arachnoid, spaces; the tunica-vaginalis; the joints; and the eye-ball, especially the aqueous chambers. But in the last three situations dropsy is excessively rare.

Cardiac anasarca *commences* almost invariably about the ankles and feet, gradually extending upwards: still I have known it originate about the eyelids, where the kidneys were functionally and texturally healthy; such cases, excessively rare, belong to the class of dilatations with tricuspid regurgitation. When carried to great amount, erythema, erysipelas, and even sloughing are prone to occur: the skin cracks, and through the fissures a fluid, at first glutinous, afterwards watery, oozes more or less copiously.

The various sites enumerated may, for practical purposes, be grouped under two headings, *systemic* and *visceral*.

SYSTEMIC FORM OF CARDIAC DROPSY.

511. Dilatation was formerly regarded as the condition of the heart mainly inducing the important class of dropsical symptoms. This opinion, discountenanced by Bouillaud, who sought to establish valvular obstruction as their sole efficient cause, was restored to favour by Hope and Andral,—the former of whom, indeed, went the length of teaching that pure hypertrophy, also,

was capable of generating dropsy. Of late, Blakiston has brought together a body of evidence calculated to show that the systemic vessels do not become loaded in cases of dilatation, unless there be coexistent tricuspid regurgitation. Still more recently H. Douglas has defended the original thesis of our forefathers.

512. It becomes necessary for me here to express an opinion on this "vexed question" of the mechanism of cardiac dropsy; let me commence by throwing into a series of propositions such inferences as flow directly from facts that have fallen under my own notice.

(*a*) 1. Mitral regurgitation or obstruction, or aortic regurgitation or obstruction, may severally exist, and, for a lengthened period, without systemic dropsy supervening. 2. Mitral regurgitation and aortic regurgitation may coexist for years, and yet no dropsy occur. 3. Both of these propositions (1 and 2) hold good, whether notable hypertrophy do, or do not, exist behind, or in connection with, the obstruction. 4. Simple hypertrophy of the left ventricle may reach the highest point without systemic congestive effects of any kind arising. Hope, as is well known, maintained the reverse, holding that pure hypertrophy, if protracted, will produce general dropsy; but he gives no cases demonstrating the fact, and the motives of his belief are, as far as he shows, totally speculative. And *à priori* views are not so completely in his favour as he appears to imagine; when he talks of the "increased force of circulation surmounting the natural tonic power of the capillaries," he forgets, that very power, as well as the nutrition of the vascular walls, *may* have increased *pari passu* with, and in consequence of, the growth of the cardiac hypertrophy [493, *b*]. The question is one of observation; and I have stated what I have actually seen in persons who had not been reduced by treatment or otherwise to a state of anæmia. 5. Dilated hypertrophy, even, of the left ventricle, may last for years without any such effect ensuing, provided the dilatation be not in notable excess. 6. The heart may be in a state of advanced fatty metamorphosis, the pulse feeble and infrequent, the encephalic and respiratory functions exhibit the singular perversions attending a high degree of that disease, the entire organism betray functional languor and inactivity, and yet even the prætibial integuments fail to pit in the least under pressure. 7. Or the heart may be soft and flaccid, and the pulse persistently frequent, feeble, and irregular in force and rhythm, and yet no systemic congestions occur. 8. The natural relationship of width of the arterial orifices, and also of the auriculo-ventricular orifices, may be materially perverted, without the least systemic dropsy arising, until the closing days of life. This is seen, for instance, in cyanotic cases where the aorta and pulmonary artery are transposed in origin, the relationship of the great veins to the two sides being normal.[*] Similar evidence is often afforded by cases of aneurism of the arch of the aorta, where the orifice of that vessel is more or less widely dilated, so as to exceed in width that of the pulmonary artery to variable amounts. 9. Tricuspid regurgitation, where the right ventricle is in a state of dilated hypertrophy, as shown during life, by swollen and pulsatile jugular veins which fill from below, and as shown after death by actual examination, does not necessarily produce dropsy.[†] Besides if tricuspid regurgitation sufficed, unassisted, on mechanical principles to produce systemic dropsy, why should not constriction of the pulmonary orifice, so frequent in cyanosis, habitually engender it?

(*b*) These propositions are, I believe, incontrovertible; they are the mere general expression of facts which are perpetually occurring. I cannot, then, see how the conclusion is to be avoided, that something beyond and in addition to any one, or any group, of the cardiac conditions referred to, is required in order, as matter of necessity, to entail the occurrence of dropsy. I can scarcely suppose the unwarranted assumption that, were life sufficiently prolonged, those conditions would of themselves suffice for the purpose, would be seriously urged in rejoinder. And again, the existence of some active cause beyond, and independent of, the heart, is further shown by the facts: that there is no direct relationship between the amount of heart-disease and of dropsy; that dropsy comes on suddenly sometimes from extraneous causes, the state of the heart remaining, as far as ascertainable, in precisely its previous condition; and that dropsy diminishes and increases, comes and goes, either spontaneously or through the influence of treatment, while the organic changes in the heart remain permanent and unmodified.

(*c*) We must not, however, run into the opposite and equally erroneous extreme of wholly ignoring the direct influence of organic changes of the heart and its orifices. Two cardiac affections are, as matter of experience, frequently associated with systemic dropsy,—namely, dilatation and tricuspid regurgitation. And it is certainly so rare for either of these states to exist for any length of time without the supervention of such dropsy, that any hypothesis, explanatory of its occurrence, must look to these states as forming important links in the chain of causes.

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[*] Medico-Chir. Transactions, vol. xxv. p. 1, 1842.

[†] W. Hallington, ætat. 53, died at U. C. H., April 7, 1849, with fatty dilated enlargement of the heart (weight 17½ oz.), dilatation of the tricuspid and mitral orifices, and aortic regurgitation, all three carried to a great extent, and yet not a particle even of œdema about the ankles had appeared. The patient had also an aneurism of the abdominal aorta, at its bifurcation; but this could not be held to be *preventive* of dropsy.

(*d*) A share in the direct mechanism of systemic dropsy being thus conceded to structural change in the heart, the question next arises what is the nature of the influence, independent of that organ, which completes the causation. Local conditions in the heart, we admit, establish a difficulty in the systemic circulation; what influence actually and directly leads to the dropsical exosmosis?

This influence seems a compound of conditions, favourable to transudation of the serosity of the blood, in that fluid itself, in the walls of the capillaries and venous radicles, and in the receiving tissues. *First*, as concerns the blood, the influence of an impoverished state of that fluid is too well known to be for a moment contestable. Experiments on animals prove that if the veins be more or less loaded with water, they yield this in the form of dropsical effusion. The œdema, and sometimes extensive anasarca, of spanæmia; the slight œdema attending the hypinosis of protracted convalescence from various acute diseases; the various dropsies of uræmia,—are all illustrations in point. Obviously morbid states of the blood, when of the fitting kind, of themselves alone suffice for the production of dropsy: look at the sudden anasarca of acute "Bright's disease," or of an acute recrudescence in the chronic affection, while the heart, liver and lungs, may be texturally sound. *Secondly*, it is readily conceivable that the variable density of texture of the walls of the vessels shall promote or restrain the process of filtration. *Thirdly*, cases occasionally present themselves, in which dropsy, supervening from diseased heart, fails to affect portions of the body, noted, under ordinary circumstances, as the earliest and readiest sufferers,—for instance, the lower extremities. I have observed this, where the legs had been the seat of erysipelas and subcutaneous inflammation prior to the occurrence of the cardiac dropsy: the chronic anatomical changes in the connective tissue in such a case possibly act as a barrier to its reception of serosity from the vessels.*

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* Clin. Lect., case of Hope, Lancet, 1849, vol. i., p. 442. Robertson, U. C. H., Females, vol. viii., p. 295.

It may no doubt be argued *à priori* that dilatation of the heart, occurring as a primitive disease through simple weakness, or following actual structural alteration of the texture of the organ, will occasionally prove the efficient cause of dropsy, even admitting the necessity of blood-change just contended for. It may be urged that, when the heart is so affected, the necessary *vis à tergo* in the circulation is wanting, and capillary stagnation must ensue, —and that this very stagnation, becoming habitual, may modify the qualities of the blood, and impair the nutrition of the walls of the vessels through the strain they suffer. But plausible though this argument be, I believe, as matter of experience, that the necessary change in the composition of the blood is meanwhile really worked out by other and more effective agencies.

513. Long since, Lower showed that local dropsy might be caused by ligature of veins; and the occurrence of serous effusion from local obstruction of vessels of that class is clinically well known. But here are instances of *sudden* and *local* obstruction; they are evidently not logically comparable with cases of *slow* and *centric* obstruction. And even where the difficulty in the way of the returning blood is purely local, it does not necessarily follow that transudation shall occur from the communicating venous radicles, although the evidences of changed condition of those vessels be structurally obvious. Thus in cases of aortic aneurism, pressing on one or both innominate veins, the capillaries of the base of the neck may dilate in such manner, as to produce notable general swelling at the spot, yet not a particle of œdema be discernible.

514. *Treatment.*—The treatment of cardiac systemic dropsy must vary somewhat with the precise conditions of the heart; but there are some points of general applicability which may here be set down. There is no period of a given cardiac disease at which the removal of dropsy may not be accomplished; but when the powers of the patient are seriously enfeebled, great caution, as regards the rapidity of that removal, is called for.

(*a*) The dropsies of heart-disease are very rarely attended with true plethora, and hence on that score very rarely call for venesection. But, when a clogged condition of the heart's cavities coexists with pulmonary congestion and anasarca of more or less acute course, and the general vigour is as yet unimpaired to any serious degree, the abstraction of a small quantity of blood, either by venesection or, better, by cupping of the chest, has appeared to me to facilitate the action of hydragogue remedies. That diminished fulness of the vascular system promotes absorptive power, has been well known since the day of the convincing experiments of Magendie; but, even where the quantity of blood abstracted scarcely authorises us in resorting to this explanation, the result is beneficial.

Of hydragogue purgatives, peculiarly valuable from their certainty of action, elaterium,* gamboge, bitartrate of potass, and the pulvis jalapæ compositus are the most valuable of the class. Stimulants may be requisite during the use of these medicines; and their action may be so rapid, that the mere loss of fluid shall weaken vitally and mechanically. An ounce and a half of the bitartrate of potass may be given with perfect safety, freely diluted and combined with carminative tinctures,—smaller doses having first been employed.

Diuretics, uncertain in their effects, are often rendered much more active by preliminary cupping or dry-cupping of the renal regions. It will be necessary to vary the combination from time to time, and the list for selection is fortunately a full one,—comprising the acetate, nitrate and bitartrate of potass, the iodide and bromide of potassium,† nitric ether, the spirits and infusion of juniper, squill, colchicum, decoction of scoparium and of chimaphila, and infusion of digitalis,—or, if the liver be at all engorged, the extract and decoction of taraxacum. Of all these agents the infusion of digitalis has appeared to me the most active, and no amount of apparent weakness of the heart is *per se* an absolute contraindication to its use. Hydragogue purgatives often promote the action of more direct diuretics; and small doses of blue pill, occasionally, at bed-time, are very useful adjuvants.

Diaphoretic drugs are of little utility; but the free diaphoresis produced by the vapour, or hot-air bath, is sometimes strikingly beneficial.

If there be any tendency to bronchitis, bronchial discharge should be encouraged by the free use of expectorants.

Now there are few conditions of disease in which the effects of treatment are more striking up to a certain point than in cardiac dropsy: every now and then a patient is snatched from instant death, freed wholly from anasarca, and restored for a time to a state of fairly comfortable existence. And the process may be repeated more than once. As far as my own observation goes, these desirable results are best and oftenest obtained by the judicious use of infusion of digitalis and elaterium.

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* The following is a useful formula:—℞ Extract. Elaterii, gr. $\frac{1}{8}$—gr. $\frac{1}{4}$; Creasotonis, gt. i.; Extract. Hyoscy., gr. ii.; pro pil., i., repetend. prout opus sit. The inequality of strength of the extract, as supplied by various druggists, renders it prudent to commence with a very small dose: the greater certainty of the strength of the alkaloid, momordicine, will probably lead one day to its being generally employed, though with a purgative of such intense activity, and, at the same time, occasionally depressing character, the greatest caution must, of course, be observed.

† The iodo-bromuretted waters of Adelheidsquelle, Kreuznach, and Woodhall, act diuretically in some cases. Where iron is contra-indicated, the Lincolnshire Spa will be the most serviceable of the three.

(*b*) Removal of anasarcous fluid by mechanical means becomes necessary, if medicines fail in controlling its increase; and if inflammatory changes in the skin appear imminent. Scarification is hazardous,—being not unoften followed by erysipelas; while acupuncturation over the insteps, or at the upper and inner parts of the thighs, though a slower process, is not attended with the same danger: there is even some advantage in the slowness of discharge; too rapid evacuation has been followed by great depression. Life is occasionally preserved for months, nay, for years, after the spontaneous or artificial relief of the anasarca,—and this under the most apparently disastrous circumstances. It is scarcely necessary to add that, should discharge have commenced, either through spontaneous fissures or artificial punctures, it is extremely dangerous to arrest the flow by healing the openings: I have known death rapidly ensue from dyspnœa and thoracic dropsy under the circumstances.

The patient's strength must be supported by food containing much nutriment in a small compass; and, though thirst torment him, he must refrain from fluids as far as possible. Stimulants may be required; and hollands or gin have both popular prejudice and medical experience in their favour under the circumstances.

(*c*) Lastly, we from time to time meet with examples of cardiac dropsy which resist treatment more or less completely, and disappear spontaneously. The rationale of the disappearance commonly baffles detection: the worn out explanatory formula, "improvement of the general health," completely fails us.

VISCERAL AND INTRA-SEROUS FORM OF CARDIAC DROPSY.

515. *Pulmonary œdema* is chiefly connected with disease, stenotic or regurgitant, of the mitral orifice,—sometimes with dilatation of the left ventricle, very rarely with hypertrophy of the right. *Ascites* arises especially from general dilatation of the heart and tricuspid regurgitation; and these are the conditions mainly observed as antecedents to all the other varieties of internal

dropsy enumerated. The mechanism of hydropericardium has already been sufficiently considered [459].

B.—CARDIAC FLUX.

516. (*a*) The intestines become the seat of serous flux in some rare instances of certain forms of heart-disease. These forms are general dilatation and tricuspid insufficiency. Watery diarrhœa thus induced may be wholly spontaneous; or the original excitant of the discharge may have been some hydragogue purgative, the influence of which holds on. Unless excessive, flux of this kind is rather to be encouraged than otherwise, probably saving the patient from dropsy of the areolar tissue or of the serous sacs. But as far as my observation goes it rarely occurs in the purely spontaneous form: when it does so, its utility is much greater than when excited medicinally.

(*b*) In some of those cases where vomiting forms so obstinate a symptom of chronic heart-disease, the immediate cause of the nausea seems to be the presence of an irritating watery secretion from the gastric surface. Such vomiting is never very copious.

(*c*) Watery flux from the kidneys, though on the whole not very rare, is uncertain in its occurrence, doubtful in its mechanism, and cannot be said to belong to any particular cardiac affection. For my own part I have seen passing attacks of hydruria more frequently in connection with different forms of dynamic disturbance than with any specific organic disease. It is not very uncommon for fits of palpitation, purely functional, to terminate by, or with, a copious discharge of aqueous urine; so, too, anginal and pseudo-anginal seizures, or passing fits of mere paræsthesia of the cardiac nerves, may be followed by hydruria. I know a case of the latter class in which two and even three quarts of such urine were occasionally discharged in the twenty-four hours after the seizure, the patient being habitually remarkable for the small quantity of his renal secretion. Now, though the doctrine of eccentric pressure on the Malpighian capillaries, already referred to [491, *h*], might with some plausibility be adopted in the case of hydruria sequential to grave palpitation, I do not see the smallest *locus standi* for such explanation, when the discharge follows simple paræsthesia. Here the rationale is the same as in the case of hysterical diuresis; and so long as the immediate mechanism of the latter continues in its present obscurity, we must remain in ignorance of that of the former.

(*d*) Albuminuria occurs every now and then in cases of cardiac disease, especially of dilatation and tricuspid insufficiency, the tendency of which is to congest the viscera. The cortical and tubular substances remain texturally sound.[*] The characters of this form of albuminous flux are as follows: it is a passing phenomenon, sometimes disappearing spontaneously, sometimes yielding to treatment; the albumen is always small in amount; the specific gravity of the fluid never falls very low, its colour remains unaffected and its smell wholly free from that whey-like odour to which I long since drew attention as notably significant of "Bright's disease."[†] A few casts of the tubules occasionally appear.[‡]

IV.—DILATATION OF THE HEART.

517. (*a*) By dilatation of the heart, is understood that state in which the capacity of its cavities is increased disproportionately to the thickness of their walls. It occurs in three forms: *hypertrophous dilatation*, where dilatation predominates, but the walls are somewhat thicker than in health; *simple dilatation*, where the walls are of such thickness as would be normal, had the capacity remained unchanged; and *attenuated dilatation*, in which the walls are distinctly thinner than in health,—to such a degree that the thickness of the wall of the left ventricle may, in some points, positively not reach one line. Clinically, as well as anatomically, the characters of dilatation are obvious in proportion to the excess of capacity over thickness; and the description of the disease about to be given must be understood to refer to cases in which such excess is prominently marked.

(*b*) Dilatation, carried to serious amount, usually involves both ventricles; one, however, to a greater extent than the other. The disease is not uncommonly limited to the right, very rarely to the left, ventricle. The form of the dilated ventricle becomes globular, and the apex of the heart so rounded off, that it may with difficulty be identified. The transverse measurement of the organ undergoes much greater increase than the vertical. In rare instances the auricles are predominantly affected.

(*c*) Dilatation of the orifices only occurs, as a rule, where both

[*] G. Smith, U. C. H., Males, vol. xi., p. 196; also Couch, U. C. H., Females, vol. vii., pp. 341—345. But examples of the fact are of daily occurrence.

[†] Clin. Lect., Lancet, 1849, vol. i.

[‡] Just as they do in pyelitis, in jaundice (Scott, U. C. H., Females, vol. v., p. 93), and probably in a variety of other affections.

auricle and ventricle on the same side are implicated : insufficiency of either auriculo-ventricular valve may thus be produced independently of structural disease, or even dynamic imperfection, in itself.

(*d*) Dilatation, especially when pushed to extremes, notably impairs the activity of the blood's movement through the heart,—hence follows a tendency of variable force to accumulation of blood in the cavities. The physical signs of such accumulation may occasionally be caught during life, and after death grumous coagula are commonly found,—the tendency in question being sometimes, though rarely, counteracted by the actual mode of dissolution.

(*e*) The tissue of the dilated walls is more or less soft and flabby—and if this deficiency of firmness be disproportionately marked at any particular spot, the ventricle may there yield, undergo pouching, and a true aneurism of the heart ensue. The colour of the muscular substance may be deep brownish red, anæmically pale, or mottled yellowish brown. Microscopically, the fibre is deficient in striated character, and granular looking; fat, extra- or intra-sarcolemmous, may exist; and in some cases the appearances of adventitious cellulo-fibroid texture are traceable, the muscular substance being locally or patchily cirrhosed and atrophous.

518. *Causes and Mechanism.*—If any one of the affections of the orifices of the heart, tabulated in a former place as efficient causes of hypertrophy [487], act upon a heart of weak resistant power, and predisposed to suffer from an under-par force of nutrition, the result of the eccentric pressure of the blood on the endocardial surface will be detrusion outwards of the walls and expansion of the cavities.

But no constant ratio holds between the amount of dilatation and the apparent valvular difficulty or difficulties : and in certain cases no statical valvular mischief can be satisfactorily ascertained,—while, as far as can be judged, no dynamic disturbance has existed during life playing upon the orifices. Hence it is supposed that weakness of the heart's fibre, pure or independent of definite structural change, renders the walls of the organ incapable of resisting the eccentric strain arising under the ordinary effort of the circulation.

But, there is, besides, the influence of mechanical difficulty beyond the heart itself to be considered. Does aneurism of the aorta, especially of its arch, entail dilatation of the left heart? The affirmative has been very confidently taught, but proof of the soundness of the doctrine has never been given. I know, as matter of observed fact, that not the smallest increase of area or of bulk of the left ventricle may exist, even when a very large sac originates just above the sygmoid valves; and hence feel disposed to believe that dilated hypertrophy and aneurism, when found together, are mainly related as joint results of a common cause.

Persistent capillary obstruction in various organs is ordinarily supposed to dilate the heart in the course of time. The arguments, used in considering the power of renal obstruction to produce hypertrophy, will apply in the case of dilatation, quoad the mechanics of the question (p. 289).

The influence of all obstructive diseases of the lung of asphyxiating character in dilating the right, and eventually the left, heart, has long been known. Mere capillary obstruction and destruction will not suffice for the purpose; for as a rule the heart, instead of rising above, falls below, the par of healthy bulk in phthisis. A probable enough explanation is that in phthisis the mass of the circulating blood undergoes gradual decrease, proportional as it were to the capillary destruction within the lung. Again, it may be, that, as W. T. Gairdner suggests, enlargement of the heart takes place in collapse of the lung and emphysema, "because the expansion of the thorax in inspiration tends constantly to overload the heart." *

There still remain cases wherein dilatation, inexplicable on any of the theories now reviewed, appears traceable to some intrinsic fault in the heart itself, the nature of which remains unrevealed.

519. *Repulsions.*—It seems somewhat like "slaying the slain," to denounce the doctrine of absolute mutual repulsion between cardiac diseases, such as dilatation, attended with venosity of the blood, and active tuberculization. A single fact, elsewhere recorded, would suffice to show the theory cannot stand.†

Cases occasionally occur, telling both for and against the theory. Thus a patient may be tuberculous, and then become the subject of dilatation and tricuspid regurgitation, while pre-existing tubercle either becomes quiescent or undergoes retrograde changes: and yet the death of such a patient, whose life holds on at the time despite a persistent partial asphyxia, maintained by the heart-disease, may be in fact eventually due to the acute development of grey granulations.*

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* Brit. and For. Med. Chir. Rev., July, 1853.

† Diseases of the Lungs, ed. 4, p. 507 (*b*); also Crocott, U. C. H., Males, vol. xviii., p. 113.

520. In order as far as possible to ensure precision, it seems advisable to consider apart, dilatation involving the heart as a whole, and dilatation of particular cavities. The student will, however, bear in mind that the latter cases are rare, and of relatively small practical importance.

A.—GENERAL DILATATION.

521. *Physical signs.*—(*a*) The apex-beat is very indistinctly visible, or, if the patient be full in person, actually invisible; when seen, it may fall nearer the sternum than the natural point of the apex-beat, in consequence of the rounded-off form of the heart annulling, or lessening the clearness of definition of, the true apex; if the dilatation be considerable, the apex strikes the surface *below* the fifth interspace. The impulse, whether more extended than natural, or limited to the fourth and fifth interspaces, frequently exhibits the quasi-undulatory character. There is no prominence of the cardiac region.

(*b*) As felt by the hand, the shock is feeble in proportion to the purity of the dilatation: the apex-beat, though visible, and though movement be nowhere else visible, frequently cannot be distinguished by the finger from the impulse of the ventricles generally, —an evidence of extreme motor feebleness. The impulse, non-vibratile, either consists of a short feeble slap, followed by a sudden fall back of the organ, or of a more prolonged faint tremulous motion. The force of successive beats is unequal; their rhythm may be irregular to a slight or to the very highest degree; and there may be a want of perfect uniformity in the point of the surface struck by the heart in the successive beats of a series, quite independently of the influence of respiration. Palpitation may notably increase the force of impulse, though it often does so but very slightly.

(*c*) The *intensity* of percussion-dulness in the superficial cardiac region is not, as it probably may occasionally be in hypertrophy, increased; and in cases of attenuated dilatation the parietal resistance, it is averred, may be less marked even than in the state of health. Both these signs are, to say the least, difficult of detection and insignificant in value. The phenomenon of horizontal conduction of percussion-sound [72] must always interfere with both. The *areas* of dulness, deep and superficial, are both widened: the former inclines to squareness of outline, the transverse diameter of the heart having proportionally undergone a notably greater increase than the vertical.

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* Clin. Lect., case of Hishin, Med. Times, February, 1857.

(*d*) The systolic sound varies in characters according as the cardiac tissue is firm or soft,—a variation which is intelligible whether muscular action be, or be not, accepted as an actual part-cause of the sound.

In the first place, if the fibre be firm and fairly nourished, the heart's systolic sound, both at the base and apex-region is, generally speaking, though short and abrupt, full-toned and of higher pitch than in health. It is superficial, appearing to be produced close under the spot of chest-surface auscultted; its maximum point is generally slightly lowered. The diastolic successor is not specially affected. Both sounds, as heard at the top of the sternum, bear the natural relationship, in point of intensity and resonance, to those audible over the heart itself,—a circumstance which in occasional cases of enfeebled cardiac impulse may help the observer signally in excluding hydropericardium, as a possibly existent state.

In the second place, if, on the contrary, the dilated ventricle be soft, flabby, or fatty, the first sound may be very weak, faint, toneless, and of high pitch; and the second so feeble as to be inaudible at the apex, especially if the heart's rhythm be markedly irregular.

So, too, upon the quality and mass of the heart's texture will depend the extent to which the sounds are transmitted through and over the chest; if the dilated ventricles be well nourished, and *à fortiori* at all hypertrophous, the sounds, approaching in attributes those of hypertrophy with dilatation, will be widely diffused, and clearly audible in the right back; if, on the contrary, the dilated heart be a soft, flabby one, the area over which its sounds are audible may be extremely limited.

A slapping noise, the intensity of which may be increased by causing the patient to bend forwards, is sometimes produced by the collision of the heart against the side.

Dilatation is not habitually productive of any intra-cardiac murmur, unless the morbid state has proceeded to such lengths as to render certain valves incompetent to close their orifices: this may occur with the tricuspid, more rarely with the mitral, and still more rarely with the aortic valves. The murmurs thus engendered are, of course, always regurgitant; but they do not always occur when their physical cause, incompetency of the valves, actually exists, inasmuch as the strength of the ventricles is sometimes incapable either of giving the necessary impetus to the regurgitating stream, or of calling forth such energy of its own systolic reaction on the part of the aorta, as is required for the production of murmur. It is also possible that dilatation, by giving an unnatural direction to the blood-current, may engender murmur [106, *n*].

Are reduplications common when the ventricles are unequally dilated? I think so; but my number of observations on the point is too small to justify a positive assertion [103, *k* (2)].

522. *Symptoms.*—Few affections vary more notably in the intensity, few agree more completely in the character, of their local and general symptoms, than general dilatation of the heart. To describe all the degrees clinically observable, modified, too, as they are, by the idiosyncrasy of the individual and by the manner of complication with changes structural or functional of other parts, would prove an endless task; it will suffice to place before the reader a picture of the maximum amount of suffering the disease entails.

And it is important to remember that cases, in which dilatation of the heart exists in an isolated state symptomatically, are rare: in the first place such dilatation, as an actual pathological state, is uncommon; and, secondly, advice is scarcely sought until some of the secondary lesions make themselves felt through their own special disturbances of function.

523. Taking, then, as our imaginary model, a case of highly marked general dilatation, attended with the ordinary secondary changes which time works out, the failures of the various functions will be found of the following kind.

(*a*) The patient, habitually irritable and melancholy, breaks from time to time into fits of despondency and petulant complaint; he is deficient in energy, both mental and bodily—the dislike to movement is only overcome under the persuasion that exercise is in some measure essential; the strength fails; the weight, unless factitiously sustained by dropsies, falls very notably; sleep, in the real sense of the term, is rarely enjoyed,—fitful dozes, abruptly interrupted by startings and frightful dreams (true incubus is not more common than in persons free from heart-disease), are its nearest substitute; habitually the patient lies with his head high, and when under the pressure of a fit of dyspnœa, or "cardiac asthma," unable even to lean backwards, he bends the head forwards, or sits erect with the feet hanging out of bed; his debility and dropsical unwieldiness prevent him from assuming some of the peculiar postures in which the subject of pulmonary spasmodic asthma struggles for breath. But there are cases of well marked dilatation in which the patient habitually keeps the head low; I have known it constantly maintained on the level of the shoulders. Nay, where in addition to general dilatation of the heart there exist cardiac dyspnœa, congestion of the lungs and hydro-thorax, the sufferer may, as I have not unoften seen, be easiest with the head low. The explanation is the same as in the case of hydropericarditis (p. 225, *note*).

(*b*) Chilliness of the extremities, livid discoloration of the prominent parts of the face, mottled with patches of sallow and earthly tint, or varied by leaden or almost black discoloration about the eyes and mouth,—conditions of colour all of them most marked, as a rule, in the morning; lividity or blueness of the lower extremities generally, with excess in particular spots,—spots which, eventually falling into a state of absolutely stagnating circulation and thrombosis, slough, independently of calcification or inflammation of the arteries or veins or of embolism; and, lastly, anasarca, softly pitting, spreading from the feet to the abdominal, and even the thoracic, walls, the external genital organs, the face and neck, rarely the upper extremities,—constitute a series of conditions showing how deeply the tegumentary structures suffer.

(*c*) In the locomotive system there is little to notice. The joints remain unaffected. I have seen hæmorrhage into the sheath of the rectus abdominalis.*

(*d*) Anorexia and nausea may be habitual. The latter, indeed, occasionally becomes the dominant source of suffering,—the sense of nausea scarcely ever ceasing, while the simplest food in the smallest quantities is invariably rejected. In this state there may be no biliary derangement, and the epigastric region be perfectly free from pain or tenderness. The mechanism of the symptom probably varies in different cases,—sometimes depending on mechanical congestion of the gastric mucous membrane, sometimes on mechanical pressure by the heart from above, sometimes on peculiar secretion [516, *b*], and sometimes on reflex irritation of the vagus nerve. I have occasionally known such nausea disappear spontaneously after having resisted all kinds of apparently rational treatment for weeks together. The tongue (unless some intercurrent state exist to modify its characters) is broad, full, not always pitted at the edges, however, and of dark purplish ground,—the fauces venously injected. Hæmorrhage may occur from various parts of the alimentary mucous tract; I have seen this in the case of the bowels, when the dilatation alone seemed its direct cause, but do not feel positive that hæmorrhage from the stomach has occurred under my observation, unless there were intermediate high congestion of the liver,—the hepatic system may act as a sort of safety-valve of the stomach, until itself grows over-loaded. Hæmorrhoids are not so common as might be expected,—the bowels habitually constipated, or alternately relaxed and confined,—the discharges dark. The liver is more or less congested: the hepatic system suffers first, the portal secondly; unless both are implicated, the size of the organ is not sufficiently increased to affect the results of percussion notably: the texture of the lobules may remain perfectly sound. Positive jaundice does not occur from this state of the liver alone; but I have seen a faint yellowish tint during life, where death disclosed nothing but hepatic congestion to account for it. Ascites follows on anasarca; I have no experience of the peritonæal dropsy as a precursor of the sub-cutaneous.

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* Hishin, U. C. H., Females, vol. xi., p. 289.

(*e*) Venous stagnation, with its consequences, occurs in the lungs.—Here may be enumerated oppressed or sighing breathing, and eventually orthopnœa, complete and habitual, with occasional paroxysms of dyspnœa [642]. The respiration may actually stop when the patient dozes off,—as if the influence of the will were necessary to sustain the act. This is probably a symptom of fatal augury,—as it certainly proved in the few cases of the kind that have fallen under my notice.

But in point of fact all the varieties of cardiac dyspnœa are observed, and to the section on that form of respiratory disturbance the reader is requested to turn for further information: *vide* CARDIAC BREATHLESSNESS.

In addition cough, dry, harassing, and even convulsive, or accompanied with expectoration, serous, rarely frothy, or stained or streaked with blood, or mixed with a little blood, fluid and dark, or in pellets, increases the distress. The physical signs of bronchitis, or of pulmonary congestion, irregularly disseminated, though most marked at the posterior bases, of pulmonary œdema, or of pulmonary apoplexy, may be found,—actual hæmoptysis to large amount may accompany the latter state quite independently of mitral disease.[*] In rare instances considerable hæmoptysis occurs, where the anatomical conditions of pulmonary apoplexy are not to be found after death.[†]

(*f*) The organs of circulation themselves furnish the following symptoms. Feeble, fluttering, distressing palpitation, increased by the slightest movement, either occurring from some obvious cause, or, as frequently, without apparent exciting influence, is a standing source of misery to those sufferers. So, too, is uneasiness in the cardiac region, of characters most difficult to describe,—of an intensity varying between a mere sensation which constantly reminds patients that, as they often express themselves, "they have a heart," (the mildest form of cardiac paræsthesia) and the agony of angina,—paroxysms of which may actually occur and put an end to existence. Tenderness on pressure may be discovered over the heart in some rare cases.

The pulse is either natural in frequency, small, feeble, and abnormally posterior in time to the ventricular systole, but regular; or it is frequent, quick, narrow, feeble, short, fluttering, polycrotous, and irregular both in force and rhythm. The latter state is either limited to the periods of palpitation, or, if constant, is indicative generally of softness of the heart's texture. True intermittence is rare. The superficial pulses are not visible. Faintness, rarely lapsing into actual syncope, occurs from time to time.

The sphygmograph exhibits to the eye the various tactile characters just enumerated,—but gives no specific trustworthy diagnostic information (*vide* APPENDIX).

(*g*) Swelling of the abdominal lymphatic glands is said sometimes to take place: I have never seen it,—possibly because sufficient search was not made.

(*h*) The kidneys are passively and mechanically congested like the lungs and liver; the renal regions may be tender on deep pressure posteriorly; but I have never known the organs sufficiently increased in bulk to cause any positive extension of the area of their postero-lumbar percussion-dulness. The urine is small in quantity, high coloured, loaded or not with lithates. Albuminuria may occur from mere renal congestion,—the urine possessing the characters already described under the head of cardiac flux: [516, *d*] this symptom often disappears spontaneously, and may almost always be quickly removed by certain measures directed to the kidneys, especially cupping or dry-cupping. In rare instances spontaneous hydruria occurs temporarily; I have known most copious diuresis of the kind preceded by violent rigors, for which no cause could be detected.

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[*] Fosbury, U. C. H., Males, vol. x., p. 133; Whittaker, U. C. H., Males, vol. xviii., p. 85.

[†] The minute branches of the pulmonary artery are then probably themselves diseased.

(*i*) Sexual inclination and power, though weakened materially, are not annulled,—rupture of a dilated heart has occurred *in actu coitûs.* It is said that uterine hæmorrhage is sometimes traceable solely to dilatation of the heart: the statement, unconfirmed by any cases that have fallen under my notice, seems to stand in need of corroboration. I certainly have, nevertheless, seen some instances of profuse menstruation in females of an age more or less advanced beyond thirty,—and in whom coexisting mechanical congestion of both liver and kidneys made it probable enough, that a similar condition of the uterus might have played a part in increasing above par the quantity of catamenial discharge through the vagina.

(*k*) More or less congestion of the encephalon can scarcely be escaped, where the face exhibits marked indications of stagnating circulation. The dull cephalalgia, inability to exercise the mind, heaviness, torpor, somnolence, semi-coma, and finally, coma, that mark the progress of these cases, are thus explicable. Cerebral hæmorrhage and acute softening both stand in undetermined relationship to dilatation: unless intra-cardiac thrombosis have intervened, dilatation is not *per se* a sufficing cause of cerebral embolism [741, *a*]. No matter what the amount of mechanical congestion may have been about the face and head, it has never occurred to me to witness convulsive seizures, even distantly allied in character to the epileptic, as results of that congestion.

(*l*) Congestion of the spinal cord may be the cause of certain reflex phenomena, spasmodic cough, startings from slumber with affright, and convulsive movements of short duration on waking from sleep.

(*m*) Muscæ volitantes, luminous vision, fullness, wateriness, and injection of the eyeballs; tinnitus aurium, dulness of hearing; with deficiency of smell, and occasional epistaxis, from congestion of the Schneiderian membrane; all indicate participation on the part of the organs of sense.

Grouping the systemic and the pulmonary obstructions as two separate classes, the former have occurred under my observation almost invariably before the latter: Hope taught the converse sequence, but apparently on theoretical rather than on clinical grounds.

524. *Diagnosis.*—The diagnosis of dilatation of the heart turns mainly on the following points:—Weak action, quasi-undulatory impulse, indistinctness of the apex-beat;—increased area, with squareness of outline, of the percussion-dulness, the whole not lowered in proportion to its width;—heightened pitch, shortness, abruptness of the systolic sound, or great deficiency of tone in certain cases, with prolongation of the post-systolic silence;—peculiar character of the pulse;—and signs and symptoms of systemic and pulmonary obstruction and congestion.

(*a*) *Simple hypertrophy* is distinguished from dilatation by the forcible action of the heart; by the præcordial bulging; by its distinctly localised thrusting impulse; by the heart being lowered in proportion to the general area of dulness; by the muffled dulness and prolongation of the first sound and shortness of the first silence; by the characters of the pulse; and by the systemic signs being those rather of an excited than of a stagnating circulation. Every one of these characters differs, more or less decisively, from those of dilatation.

(*b*) *Eccentric or dilated hypertrophy*, also, has its forcible action, præcordial arching, and distinct apex-beat; the heart is lowered in the proportion of its bulk; the systolic sound is loud and powerful; and the pulse differs from that of dilatation. The only difficulty comes of the fact that, if the cavities be more increased in capacity than the walls in thickness, the systemic and pulmonary signs may incline to those of pure dilatation.

(*c*) *Mitral regurgitant* disease will, under ordinary circumstances, be distinguished from dilatation by its special systolic murmur at the left apex, and by the more forcible impulse: for some amount of hypertrophy in the vast majority of cases ensues on the valvular disease. Still there are two kinds of possible fallacy. On the one hand pure dilatation may be attended with the murmur indicative of regurgitant disease of the mitral valves, though those valves be sound. On the other hand, organic mitral regurgitation may, *pro tempore*, be murmurless. Now, the first difficulty will occur where the orifice is so much dilated that its valve, though perfectly sound, has ceased to be of sufficient size for purposes of complete closure [109]. There are no possible means of distinguishing such regurgitation from that induced by actual disease of the valve: happily, the phenomenon is very rare; and, as the other characters of dilatation must be present in a high degree, the treatment will not err. The second difficulty—that of murmurless organic regurgitation—will arise from feebleness of the backward current, itself depending on temporary general collapse, or weakness limited to the heart; if the heart's vigor improve, either spontaneously or by treatment, mitral murmur becomes audible.

(*d*) Dilatation differs from *chronic pericardial effusion* by the square outline of its dulness; by the distance from the clavicle to the spot of the apex-beat being natural or increased; by the heart's sounds being superficial, and *of about the same strength at the præcordial region as at the top of the sternum;* by the dulness not extending above the third rib; and by the total absence of friction-sound, no matter in what position the patient be placed.

525. *Prognosis.*—Habitually a most serious disease, the danger of dilatation increases directly as the excess of the capacity of the cavities over the thickness of their walls; directly, too, as the softness and flabbiness of the heart's tissue; directly, too, as the general deficiency of tone in the system and impoverishment of the blood. Once dropsy has supervened, life can with difficulty be prolonged by art beyond twelve or eighteen months.

Under certain peculiar circumstances dilatation of the cavities may be a much less grave affection. Larcher and others have shown that in pregnancy general dilatation may occur of a temporary kind; and Beau has apparently proved that in cases of cardio-thyroid exophthalmos dilatation may prove a passing occurrence.*

526. *Treatment.*—Dilatation of indubitable existence is not removable by treatment: those doubtful cases of the disease, which Hope professed to cure, bear no claim to scientific recognition. Still, though it be not, in the highest sense, a curable affection—though art cannot remove dilatation—art can render dilatation bearable and even unfelt. In conducting the treatment, the essential clinical element of the disease, weakness (whether dilatation be primary or secondary), must be constantly held in view, all debilitating measures systematically avoided, and every effort made to improve the heart's tone without exciting its irritability.

The statements made in a former place (p. 175) concerning the influence of digitalis would, if well founded, entitle us to expect aid from the drug in improving the contractile tone of a dilated heart. The clinical result is that regularized rhythm, slackened rate and increased firmness of contraction, do follow the use of the tincture in doses averaging from five to fifteen minims two or three times daily. It is essential that overdosing be avoided; and that every third or fourth day, a respite of forty-eight hours be given. It must be remembered that if slackening of the heart's rate of movement be carried too far, risk of coagulation in the right cavities surely arises. Digitalis is, further, useful in warding off dropsy, by maintaining a full flow from the kidneys.

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* J. W. Begbie, Vascular Bronchocele, p. 12, 1863.

Aconite may be given in small quantities, say, half a drop to one drop of Flemming's tincture, if extreme irritability of contraction coincide with weakness of beat. But the paralyzing influence of the drug should never be lost sight of.

Opium and belladonna are of subsidiary importance, as concerns the heart directly: but they may be required for the relief of secondary conditions.* Belladonna plasters to the præcordial region often do more harm than good, where weakness of heart is the dominant condition.

Nux vomica and strychnia deserve trial in view of their probable influence on muscular contractile force, and may be variously combined with iron, bark, quinine, biberine, and mineral acids in the general management of the case.

Should the heart be nervously excited, the various antispasmodics are indicated. The inhalation of laughing gas, amyl, chloroform or æther ought not, under any circumstances, or for any object, to be lightly permitted, though it be unquestionable that all four have been inhaled with impunity where the heart has been at once flabby and largely dilated, and anginal symptoms been associated [347].

The condition of the chylopoietic viscera must be carefully watched,—due action from the bowels ensured daily, without weakening purgation (aloetic medicines are the best), and the action of the liver, if this be sluggish, promoted by taraxacum, podophyllin and an occasional mercurial aperient.

The patient should lead a tranquil life, avoiding all excitement, but mixing in cheerful society. His exercise should be moderate, always considerably within fatigue. It does not appear to me that because a patient with dilated heart occasionally feels better able to walk after some minutes' effort, than he did at the actual start, therefore it is advisable to push pedestrian exercise as far as it can be endured. The natural history of angina would suffice to prove such strained work to be wrong. And, in truth, it seems scarcely questionable, error is frequently committed in the attempt to force patients, labouring under various diseases, to *walk*, *walk*, *walk*, in spite, often, of their own convictions that each additional walk has helped them on towards that bourne, whence no walker returns. Reference is not intended, merely, to the bygone well-known practice in a central provincial town,—but to the too indiscriminate recommendation of toilsome foot-exercise by many of the profession generally. I have known a man labouring under chronic nephritis, with alkaline urine and the general cachectic condition of that disease, directed to perform such pedestrian feats, by way of recovering his health, as he himself well knew would, on the very first day, have brought on an acute attack, severe enough in all likelihood to have confined him to bed or a sofa for a month. Not a doubt can be entertained that the lives of sufferers from cardiac disease are often put in jeopardy by the effort to exercise beyond their powers. Because, in the *physiological* order of things, it is good to walk, the inference is at once drawn that it is universally good in the *pathological* order of things. But, in point of fact, each disease has its own appropriate kind and quantity of exercise,—and that kind and that quantity are, as all truths in therapeutics, to be learned in each instance by *experience* alone. By the way, the Leamington practice once more tells the tale, "there is nothing new under the sun!" The ancient gymnastic physicians outdid their followers of the nineteenth century. Plato states that Herodicus prescribed a pedestrian excursion from Athens to Megara and back, a distance of 360 stadia, or upwards of forty miles! *

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* The influences of belladonna and opium are in some points of view so unlike, so opposed, indeed, that possibly the states of heart and circulation relieved by each, are not so really similar as they appear. See a paper by T. Anderson, on the Effects of Belladonna in Poisoning by Opium.

Very gentle use of light dumb-bells, by promoting the circulation through the upper extremities, seems to act beneficially; but caution is requisite in permitting this form of exercise.

A good nourishing animal diet, of plain, easily digestible character, is that fittest for these patients. Much drink of any kind is to be avoided; bitter beer in moderation is allowable at dinner, or, if the patient have been accustomed to take these, a glass, or at most two, of port or sherry. Dandelion coffee is a good material for breakfast.

A dry, bracing air, as a rule, agrees best. Particular circumstances, it is true, may require the patient to live in one of the very opposite characters; but, though a relaxing atmosphere may relieve an accompanying dry bronchitis, it unquestionably tends to depress still further the heart's energy. If the sufferer be still in a condition to take gentle walking exercise, it is essential hilly districts should be avoided, and at all events one protected level road secured for his daily turn. Places extremely hot and extremely cold, as well as those subject to abrupt changes of temperature, are unfit for the subject of dilated heart.

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* Cabanis, Medical Sketches, Tr. by Henderson, p. 406.

The influence of moderately high altitudes on cardiac dilatation, though presumably favourable, is yet *sub judice.*

The use of flannel next the skin is indispensable; the shower-bath may be cautiously tried, and continued or not according to its visible effects,—effects which, it has appeared to me, are, in all diseases, much under the influence, first, of idiosyncrasy, and, secondly, of the patient's previous habits in respect of ablutions. Tepid salt-water bathing, or even, when the affection is not far advanced, quiet immersion in the open sea, is useful; swimming exercise, it is scarcely necessary to say, should be absolutely avoided.

If palpitation or other form of disturbed cardiac action be traceable to an hysterical state, the usual remedies for that state should be given; if to flatulence, carminatives sometimes tranquillise the organ almost instantly.

Pulmonary congestion and sub-inflammation require dry-cupping, mustard poultices, flying blisters; and, as they are rarely active, squill and ammonia or senega internally. Should actual pulmonary inflammation occur, antimony must be employed with great caution: it is easier to depress the vital powers than to raise them.

Systemic dropsies are removable by the methods elsewhere described [513]. The fluid should not be too rapidly drawn off, supposing surgical influence becomes requisite, as the sudden loss of support on the part of the vessels may induce prostration of strength, from which the patient cannot be recovered: this proposition holds good in respect of acupuncturation of the limbs as well as of paracentesis abdominis.

Congestive enlargement of the liver is rapidly reducible in some patients by dry-cupping over the organ. Even the rest from toil and general comfort of an hospital, in the case of patients previously suffering from privation of all kinds, will occasionally free the cardiac circulation sufficiently in a few days, to lessen appreciably the antero-posterior and vertical measurements of the liver through disgorgement of its vascular system.

The treatment of Cardiac Dyspnœa is considered with the general description of CARDIAC BREATHLESSNESS.

B.—DILATATION OF PARTICULAR CAVITIES.

RIGHT VENTRICLE.

527. *Physical signs.*—Dilatation of the right ventricle in particular is signified by extension of percussion-dulness to the right, by excess of epigastric pulsation, by jugular turgescence, and (if there be either tricuspid regurgitation, or if some muscular strength be retained by the wall of the ventricle) by jugular pulsation of cardiac rhythm and refilling of the emptied jugular veins from below. Further, the chances of coagulation of the blood occurring within the cavity, especially when the walls are notably weak, are always to be borne in mind; systolic murmur, following the course of the pulmonary artery, may thus be generated.

528. *Symptoms.*—It is difficult to affiliate to dilatation of this ventricle any symptoms that may peculiarly depend upon it,—so frequently does some amount of dilatation of the left ventricle or some form of valvular impediment exist to complicate the problem. Theoretically, the signs of deficient oxygenation of the blood will be marked in proportion to the amount of pure or attenuated dilatation of this ventricle,—as will, also, the congestive influences on the brain and abdominal viscera. Whatever argument may be urged as to the power of unassisted hypertrophy of the right ventricle to produce hæmoptysis, it is very positive, dilatation of the cavity will exercise no such influence.

AURICLES.

529. *Physical signs.*—Direct physical evidence of dilatation of either auricle is only to be had by percussion in the natural sites of those cavities. When the jugular veins are permanently dilated and knotty, the existence of dilatation of the right auricle is inferrible, almost as matter of necessity; when the evidences of mitral disease have been unmistakeably developed for any length of time, a similar inference applies to the left auricle.

It would appear from a case observed by W. O. Markham,* that in cases of extensive extreme attenuated dilatation of the heart especially affecting both auricles, thrill, loud murmur, and pulsation, synchronous with the auricular diastole, may occur in the fifth interspace considerably to the right of the sternum,—physical signs which the observer suggests may have depended on the rush of blood from the venæ cavæ into the auricle.

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* Proceed. Med. Chir. Soc., vol. i.

530. *Symptoms.*—No special symptom can be assigned to dilatation of one or the other auricle. But the influence, a dilated auricle exercises in increasing the amount of disturbance caused by augmented capacity of the connected ventricle, is very manifest.

V.—Alterations of Consistence.

A.—SOFTENING.

531. *Nature and conditions of origin.*—Diminished consistence of the muscular structure of the heart forms an important physical element of a variety of complex morbid states of the organ. Thus the fibre loses consistence in carditis, in pericarditis, in endocarditis (at least in the strata adjoining the inflamed membrane), in hæmorrhagic infiltration, in œdema, in fatty infiltration and in fatty metamorphosis, in pure dilatation, and in certain forms of atrophy. So, too, the tissue loses firmness in certain diathetic diseases of the blood-class, in which the quality of the fibrine, whatever may be said of its quantity, undergoes impairment,—as, for instance, in cyanosis, scurvy, purpura, leucohæmia, glycohæmia, and acute phthisis. Lastly, more or less marked softness of the heart, apparently arising out of weakened innervation, as well as the altered quality of the circulating blood, occurs in variola, scarlatina, typhoid (Peyerian), and especially typhus fevers: acute fatty necrosis of fibre appears to take place in some of these cases.

Now in all these instances, softening constitutes an accident of the primary affection; but there are other cases in which the heart notably loses its consistence, without there being any prominent blood-disease, or any obvious affection of the organ itself, except the deficiency of firmness. To the latter cases alone it is proposed at present to refer: they are, doubtless, often connected with fatty metamorphosis of fibre, but they are not always so. Thus, I somewhile since saw with G. Harley, a gentleman, aged fifty-seven, of profoundly cachectic aspect, labouring under chronic pleuritic effusion, and excessively feeble cardiac action,—the impulse being invisible, and the cardiac sounds faint to a degree, the accent at the apex inclining to the second of the two. These conditions led me to surmise the existence of fatty metamorphosis, The heart, however, as ascertained, *post-mortem*, by G. Harley, proved free from that special charge, but had undergone true softening of primary and independent character. The naked-eye and microscopical appearances of the organ tallied closely with those in the case of an infant sixteen days old, recently published by E. Wagner. "The left ventricle was so soft that, on attempting to open it, it broke in pieces, and became a pulpy mass. The right ventricle was in a similar, or even softer, condition, but the auricle was normal. On putting some of the muscular tissue of left ventricle under the microscope, it fell to pieces so readily that it did not require to be teased out—fragments of muscular fibres were seen, resembling the smooth, spindle-shaped cells of involuntary muscle. They possessed no striæ, and were, for the most part, nucleated. Free cells and granules were also observed, but no fat or globules. The sarcolemma was in some parts, thickened and œdematous. The substance of the right ventricle had a somewhat similar appearance to that found in the left. No striated fibres were anywhere to be seen."*

532. *Physical signs.*—The impulse of a heart, thus variably reduced in consistence, may be persistently invisible, or visible with some and invisible with other beats, and occasionally somewhat undulatory. If any single beat be of considerable force, the left ventricle is very probably somewhat hypertrophous as well as soft. Percussion discloses nothing, unless, as is often the fact, the flaccid organ be dilated also. The first sound is short, flapping, weak, toneless, almost to extinction; the second weak and thin: there is no murmur.

The pulse, irregular in force, may be excessively so in rhythm,—no connection of time being traceable between it and the systoles of the heart. Long since Louis showed that the sudden supervention of these characters in the pulse of patients, labouring under typhoid (Peyerian) fever, was distinctly significant of an acute failure of consistence in the organ.† But though, when acutely produced under circumstances of pyrexia, such as these, softening is invariably attended with increased frequency of pulse, where the change is slowly effected by a chronic process, the pulse, though more or less feeble, fluttering, and small, is not of necessity very frequent,—the frequency may oscillate a little on either side of the par of health. There occur every now and then cases in which the beat ranges very materially below the average, —an infrequency which, though occasionally merely a deception, in consequence of weak systoles not impressing the distant vessels, is not always to be thus explained.

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* Year Book, for 1860, p. 31.

† Fièvre Typhoïde, t. ii., p. 274, ed. i., 1829. The observations of W. Stokes on the state of the heart in "continued fever" are also full of interest.

537. *Symptoms.*—There are few symptoms that can be distinguished from those of dilatation. General languor, weariness, incapacity for exertion of any kind, failure of strength, sallowness, and lividity of the face, feeble palpitation, sighing respiration, protracted fits of dyspnœa, and painful sensations about the heart, occasionally actual angina, are common to the two affections. Dropsical effusions probably do not occur, unless there be dilatation in addition to alteration of the blood: but the fact is, softening of any duration is rarely unaccompanied with some increase, general or local, of capacity of the cavities.

533. *Prognosis.*—Softening is always a serious condition: it aggravates the ill effects of any other heart-affection it may coexist with, and has of itself proved suddenly fatal, with or without symptoms of angina.

534. *Diagnosis.*—The feebleness of impulse, without increased area of dulness, and the feeble irregular pulse, without the signs of valvular, especially of mitral, disease, are the points on which the diagnosis of softening turns; but these characters will not distinguish simple from fatty softening,—and scarcely from dilatation.

535. *Treatment.*—In the management of the disease, an attempt must be made to improve the nutrition of the heart by raising the powers of the system generally, and improving the blood. Animal food, port wine in moderation, pure bracing air, regular but very gentle exercise, and attention to the state of the skin, are the hygienic means indicated. To tonics, quinine, iron, and mineral acids, we must mainly trust among medicinal agents. Astringents, provided any ill influence on the bowels can be obviated, might be advisable—among the number, gallic and tannic acids.

Quietude, mental and bodily, is indispensable; too much exercise is infinitely worse than none at all; indeed, the patient's instinct, as well as his feebleness, leads him to disobey the instructions, sometimes given in such cases, for forced pedestrian exercise. Fits of passion should be studiously avoided. Direct sedatives of the heart cannot be given without danger; and inhalation of æther or chloroform is scarcely permissible.

B.—INDURATION.

536. I have never met with a satisfactory example of increased hardness of the muscular structure of the heart, unattended with other textural change. Even in cases of hypertrophy, with hardness, this character belongs, we have seen, not to the actual sarcous structure in itself, but arises from the close packing of fibres of ordinary consistence [486]. I do not precisely understand to what condition Laennec's description of induration of the heart refers. Otto, speaking of "general inflammatory hardening," clearly designates infiltration with induration-matter.

537. The essential causes, and they rarely come into play, of induration, are contractile inspissation of solidified lymph, and calcification. Even these conditions are practically known in the local form only.

VI.—Adventitious Products.

Class I.—Non-Plastic Products or Precipitates.

A.—CALCIFICATION.

538. Precipitation of calcareous salts may occur in any one of the tissues of the heart, as well as in the actual substance of fibrinous coagula, of atheroma, of tubercle, and of induration-matter seated in or upon those tissues.

Including all these possible seats of precipitation, specimens of all varieties of form in the saline materials may be found,—granular, nodular, lamellar, and others. In the lamellar form, whether implicating the valves or the pericardium, it is that the imitation of ossification is closest, but neither histologically nor chemically is true bone ever formed.

539. The effects of calcification vary of course with its seat. In the valves it produces obstruction and regurgitation; in the cavities its effects coincide with those of polypoid blood-concretions; in the pericardium, and still more in the heart's substance, calcification, in proportion to its extent, obstructs the heart's movements; in the coronary arteries, especially if carried to any extent, it must interfere with the nutrition of the organ, and bears a certain, though as yet ill-defined, relationship to the phenomena of true angina.

540. *Diagnosis.*—I do not know of any sure means of proving that calcification exists in any one of the positions enumerated.

The existence of adventitious substance is in many of them of course easily demonstrable,—but to say that such substance is or is not in part, or wholly, calcareous exceeds our powers. Certainly we possess no means of distinguishing calcification of the endocardium from other morbid changes, interfering organically with the play of the valves. Again, it is *à priori* conceivable that calcified induration-matter in the pericardium should give an osteal quality to the percussion-note over the heart, and impress something of a similar character on the systolic sound,—conditions which, with a previous history of pericarditis, might give an inkling of the existence of the state in question. But in actual practice I doubt much whether such signs will ever be found. In a recent remarkable case,* where pericarditis was known to have previously existed, the only unusual physical condition noted during life was a slight, single, short, commonly systolic rubbing sound; this was referred to the attrition of the material of pericardial adhesion. But certainly I was unprepared for that which the *post-mortem* examination revealed as its cause,—namely, a ring of pseudo-osseous calcification in old pericardial exudation-matter, an inch broad and about two lines thick, surrounding the heart at the base.

B.—FATTY PRECIPITATION.

541. Precipitation of fat occurs in connection with the heart under two conditions essentially distinct in nature. In the one fat forms in excess in the connective tissue of different parts of the organ,—local obesity results; in the other, precipitation primarily occurs within the sarcolemma of the heart's fibre. The former condition may be distinguished as fatty infiltration, the latter as fatty metamorphosis.

I.—FATTY INFILTRATION.

542. Infiltration of fat occurs in the connective tissue in three different positions,—under the pericardium, amid the muscular fibres, and under the endocardium.

SUB-PERICARDIAL AND INTER-MUSCULAR FATTY INFILTRATION.

543. Sub-pericardial fat chiefly gathers about the base and the right side of the organ, and, no matter on which side, on the

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* Jones, U. C. H., Males, vol. xvii. p. 315.

ventricle rather than the auricle. When abundant, it encroaches on, and to a greater or less depth renders the muscular texture beneath soft and atrophous. But this influence on the nutrition of the fibres is produced by simple pressure, not by intrinsic deterioration of the sarcous elements themselves.

544. In this condition there is not a mere precipitation of oil from the blood, but an attempt, more or less complete, at the production of adipose pseudo-tissue.

545. *Symptoms.*—Laennec maintained that this affection "must exist in a very great degree before it gives rise to any serious complaint:" and he was in all likelihood right. True, cases have been recorded in some number, where the gravest effects have been assigned to such fatty disease; but very probably it must either in these instances have led to total destruction of fibre in limited places, or intra-sarcolemmous fatty change must have coexisted. There are no recorded proofs, as far as I know, that rupture of a fatty heart has ever taken place, unless where the latter condition was present; though the accident very conceivably may have occurred independently of this.

Still fatty accumulation under the pericardium produces minor annoyances, and some physical signs. Thus, I repeatedly substantiated the existence of the following symptoms and signs in the only person, a male, aged sixty-four, I happen to have long watched professionally during life and opened after death, whose heart was at once loaded with sub-pericardial fat, and positively free from serious softening or notable amount of intra-sarcolemmous oil. Sensation of oppression, or even pain, about the præcordial region; syncopal feelings on exertion; inability to walk quickly on level ground and to get up-hill, except with great and painful effort; inclination to coldness in the extremities; feeble (but, as far as I actually observed, regular) pulse, of about medium frequency; sluggish action of the liver and bowels; occasional giddiness; and feeble cardiac impulse, with a too extensive dulness under percussion, the sounds, especially the first, being weak and toneless. There was in this instance a considerable quantity of fat in the lower part of the mediastinum, which may have contributed to weaken the heart's shock against the side. Hope speaks of the pulse being irregular in such cases; but in the only instance where he made a *post-mortem* examination, the tissue of the organ was softened as well as loaded with fat. The case, above referred to, proves that irregularity of pulse is, at the least, not a necessary attendant on the disease.

SUB-ENDOCARDIAL FATTY INFILTRATION.

546. Minute pellets of fat are sometimes seen under the endocardium: in the present state of knowledge they are devoid of clinical interest. I have also frequently noticed, microscopically, fat, granular and oily, between the laminæ of the aortic valves; and also in the substance of fibrinous vegetations on the free surface of various valves; but the clinical import, if any, of these conditions has yet to be worked out.

II.—FATTY OR OILY METAMORPHOSIS.

547. Fatty metamorphosis of the muscular fibre of the heart, oil being precipitated, in lieu of wasted sarcous elements, within the sarcolemma, is an affection of much deeper interest and importance than that we have just briefly discussed.

548. The knowledge that voluntary muscle may be to all appearance converted into fat, is at least as old as the days of Aristotle. Corvisart, however, was the first to teach that the heart might undergo a similar change, and (though he does not appear to have actually seen the disease) to insist on the importance of distinguishing fatty "transformation" from fatty accumulation amid the fibres.* Laennec, in turn, not only actually saw, but so clearly described, the distinction between this form of destruction and mere fatty infiltration, with muscular wasting produced by pressure, that to his narrative of the naked-eye appearances modern information adds but little.

549. *Anatomical characters.*—The metamorphosed tissue is more or less deficient in consistence, tearing in extreme cases with the greatest ease; varies in tint from a delicate faded-leaf hue to a pale dirty-brown, mottled with darker spots; and rarely feels greasy, though readily yielding oil under pressure. The organ, in most instances natural in size, may be hypertrophous, or, as is much more common, dilated; locally infiltrated with blood, aneurismal or actually ruptured. The coronary arteries may be atheromatous, calcified and obstructed, or healthy; nor does there exist any *necessary* relationship between morbid conditions of these vessels and the muscular waste, however probable such relationship may *à priori* appear. The aortic or mitral valves, or both, are sometimes diseased; but, again, there does not seem, from what has fallen under my notice, to be any necessary, or

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* Maladies du Cœur, 3rd edition, 1818, p. 180.

even close connection between these two states; neither does calcification of the aorta appear to be more frequent than in persons of the same age, cut off without this form of fattily diseased heart.

Both ventricles are most commonly affected; the left alone more frequently than the right alone. The layers of substance immediately beneath the endocardium and pericardium commonly, but by no means always, suffer earlier than the intervening strata. The diseased change may be very distinctly limited to particular layers or islets of the ventricular substance for a variable length of time; eventually the entire thickness of the walls, papillary muscles, and columnæ carneæ, become involved. The auricles seem very little prone to this morbid change: E. Ormerod questions its ever occurring there; but I have most certainly observed it in the right auricle.

The microscopical characters of the disease are closely the same in the heart as in voluntary muscles. But its very earliest evidences are perhaps less easily caught; because, as E. Ormerod observes—an observation in which I concur—the transverse striæ are in the normal state less distinctly marked in the cardiac than in the voluntary fibre: this, at least, I hold to be true of the adult heart. Be this as it will, such striated arrangement, as normally exists, loses its clearness of definition,—the actual sarcous substance gives place to fat granules and to oil globules, which granules and globules, at first disposed in more or less evenly transverse rows, eventually lose all regularity of arrangement, and seem to take the place molecule by molecule of that substance. The nuclei of the fibres earliest disappear. The addition of æther, which dissolves the oil (an addition imperatively required for the sure distinction of the granules and globules from saline particles and from starch), will disclose elements of fibre still remaining intact in spots, where heaped-up granules had rendered them invisible. The diseased fibres are not notably, if at all, less in diameter than healthy ones.

Oil is also found outside the sarcolemma, probably in part originally precipitated there, in part reaching that situation from within the sarcolemma, either through ruptures from over-distension or through perforations resulting from atrophous absorption. As a rule, it must be remembered, the sarcolemma continues discernible, and hence the outline of the fibre is preserved; but where the destructive process has reached the ultimate point possible, this membrane itself disappears, whether by mere atrophous absorption or by fat-conversion is not clear.

Fatty metamorphosis of voluntary muscle is accompanied with accumulation of oil within the neurilemma of its supplying nerves; whether the nervous structure of the heart undergoes a similar change is imperfectly known.

550. Little advance has been made in the local chemistry of the disease. H. Weber comes to the startling conclusion that there is less fat chemically discoverable in heart-substance to all appearance fattily disorganised, than in healthy texture, in the ratio of 1·9 : 3.

551. *Coexistences.*—Fatty metamorphosis has been found in hearts of which either the external or internal membrane, or both, had been the subject of former inflammation. There seems fair reason to suppose the adjacent inflammation-process may in such cases have really promoted the mal-nutrition of the muscular substance. It is unlikely the nutrient processes could maintain their par of health, while textures so closely connected were passing through exudative and other changes. And more than this, as we have already seen [372], fatty change does sometimes appear to occur as a dependence on acute pericarditis,—and this under circumstances apparently proving the non-existence of any prior chronic affection of the heart.

In fatty metamorphosis of the diaphragm, as originally shown by G. W. Callender, the heart is almost invariably, if not actually always, similarly affected.*

But beyond this I know of no local or diathetic state that experience specially connects with fatty disorganisation of the heart. All varieties of acute and chronic pulmonary inflammation, empyema, and emphysema of the lungs may be found in cases of the disease,—there is no nexus of causality in one direction or the other. I have seen the disease in association with hepatic cirrhosis, with faded-leaf fatty liver, with true lardaceous or amylaceous and cholesteric liver, and with gall-stones,—but it is almost needless to add, the heart may be sound in each instance. The frequency of the disease in phthisis remains to be calculated; but it is not great: my own experience does not furnish me with a single example of advancing phthisis coexistent with fatty metamorphosis of the heart carried to an extent to produce notable symptoms. Chronic colourless softening of the brain, as also of the spinal cord—acknowledged effects of mal-nutrition—are sometimes found in association with it.

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* *Vide* Dis. of Lungs, 4th ed., p. 188.

Again, persons affected with "Bright's disease," scurvy, purpura, gout, chronic rheumatism (without inflammation of the cardiac membranes), and the hæmorrhagic diathesis, occasionally suffer from the affection; but, with the exception of gout, there is not a single one of these diathetic diseases that has attracted my observation by frequent coexistence. True, some of them are rarely witnessed in London practice. Nor, as far as my reading goes, has that singular diathetic disease of muscle, Cruveilhier's "progressive atrophy," been proved to involve the heart,—though is is certainly erroneous to speak of the affection as purely appertaining to voluntary muscle, seeing that it attacks the diaphragm [481]. Fatty metamorphosis is known to occur acutely in some cases of typhous cardiac softening, but not necessarily [531].

On the whole there seems to be no diathesis which positively promotes or antagonises this structural change; nor, *per contra*, does fatty degeneration in the heart clearly encourage, or exclude, the development of any other form of disease: it may prove, death occurring at an advanced age, the sole textural defect of practical significance in the body. Although sometimes combined with fatty infiltration, there is no necessary connection between the two things; rather the reverse indeed. And, further, general corpulence with local obesity of the heart may exist in the highest degree, unaccompanied by true fatty metamorphosis; while in thin, almost emaciated frames, the specific fatty deterioration of fibre is occasionally to be found.

552. *Intimate nature and mechanism.*—It would be foreign to the purposes of a purely practical work to enter upon a full discussion of the mechanism of fatty degeneration,—as exposed variously by Paget, Ormerod, Quain, Lehmann, Valentin, Handfield Jones, and others. But it may be as well to suggest that the recognized laws of vital processes, as well as attainable positive evidence, are repugnant to the admission of the direct metamorphosis of protinaceous into fatty matters in the midst of a tissue living and active,—as of so much dead material into adipocere. They who write concerning saponification of the heart as a conversion easy of accomplishment, should study the observations of Orfila,* whose exhumations prove that even in the dead tissues the process is one requiring very considerable time, and a number of favouring conditions. In a body which I, some years

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* Exhumations Juridiques.

ago, carefully examined for legal purposes, in association with John E. Erichsen, no semblance of fat-conversion had taken place, though the exhumation and examination did not take place till six months after burial. The soil in which the corpse had lain was, however, particularly dry.

It would be rash and unwarrantable on the part of any one unversed in the mysteries of organic chemistry, to deny the possibility of occasional direct chemical conversion of effete devitalised myoline into oil; but we may, I think, without presumptuousness, assume that such form of change is not usual, and that Nature's ordinary mode of procedure is this:—first, atrophy of the sarcous elements occurs, and secondly, oil is molecularly laid down in the place of the absorbed elements. The fat, according to this view, would be the mere index, in no wise the essence, of the disease.*

If so, the true problem becomes the determination of the causes of the primary failure of nutrition of the sarcous structure: and of this we are utterly ignorant. Analogy helps us little. Thus the fatty pseudo-metamorphosis of muscles adjoining non-reduced dislocations and ankyloses, and that occurring in paralysed limbs, and in bed-ridden people, is explicable by the waste consequent on inaction: but it is difficult to admit even the minor influence of any such cause in the case of the heart. Nor, again, does the fatty pseudo-metamorphosis of uterine tissue, which follows parturition, lend aid in the explanation. Now, direct evidence, as far as it goes, deposes against the merely local mechanism of the cardiac decay; there is no necessary relationship between a diseased state of the minute vessels or nerves of the heart and its failing nutrition. On the other hand, we are unacquainted with any form of blood-disease or nervous dyscrasia even seemingly essential to the existence of the disease. Smith, of Dublin, it is true, in some cases of the kind, found oil in the blood; but can we, with Stokes, accept the fact as an "apparent proof" that fatty heart may be produced by the assimilation of ready-formed oil in the blood? I am disposed to think not: the two conditions were probably mere coincidences. Simon's piarhæmia may exist without fatty heart, and fatty heart without that diseased state of the blood. And admitting the reality of some, as yet undiscovered, preliminary morbid state of the blood, it would remain to be ascertained, why that particular form of pravity of the fluid should lead to impairment of the nutrition of the heart rather than of other textures.

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* The observations of Voit and Bauer on the fatty degeneration of the organs produced by phosphorus, even in animals deprived of food and extremely emaciated, show how much remains to be learned on this obscure part of animal chemistry. (Brit. Med. Journal, May, 1872.)

Clinically, fatty pseudo-metamorphosis is closely allied to fatty infiltration of the connective tissue, along with, as its effect, pressure-atrophy of the intervening muscular fibre; pathologically, there is this difference among others, that in the latter, fat-formation is primary, sarcous atrophy its effect; in the former, the myoline primarily decays, and oil is laid down in its place. This at least I believe to be the general truth; but doubtless, as ably argued by Handfield Jones,* in some cases this distinction does not hold,—the formation of extra-sarcolemmous fat being itself the consequence of initiative atrophy of fibre.

Again, cardiac anæmia has pretty frequently (allowing for the absolute rarity of the two things) fallen under my notice as the immediate prelude to fatty metamorphosis [356]. Since I noticed this connection in the edition of 1861, a few additional instances in point have presented themselves.

553. *Predisposing causes.*—Although fatty decay of the heart has occasionally been seen in young persons, the disease is essentially an appanage of middle and advanced life. Males exhibit it, both in its slighter and graver degrees, with considerably more frequency than females. It occurs in all ranks of society, though, to a certain but undetermined extent, more in the upper and middle classes than among those who earn their daily bread by manual toil. The influence of special kinds of trade or occupation, if any, has yet to be discovered.

Aristotle ascribed conversion of voluntary muscle into fat to excess of nourishment; modern observers add the abuse of alcoholic fluids, lazy habits, and luxurious living generally, as at least favouring the metamorphosis. But I doubt very much from my own observation whether these conditions can be fairly held to play any very notable part in the instance of the heart. True, I have seen the disease in persons of sedentary, luxurious life; but I have met it also in professed pedestrian tourists, and in persons habitually and actively employed. I have seen it in men whose rule for years had been to consume at least their daily bottle of wine, in gross beer-drinkers, and in spirit-drinkers; but I have observed it also in persons whose life had been one, not only of soberness, but almost of abstinence.

Yet more, there is no doubt that fatty disintegration sometimes affects a very considerable area of the hearts of men, who at, and long prior to, the time of death, have systematically lived after the most approved hygienic models. All this forces on us the conviction that in this, as in most chronic disorganising diseases, the real predisposing cause is a certain *constitutional aptitude*, formed out of intrinsic peculiarities, congenital or acquired, of structure and function, that lie beyond the pale of our existing powers of scrutiny.

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* Fatty Degeneration, Brit. and For. Med. Chir. Rev., April, 1853, p. 334.

554. *Clinical conditions.*—The clinical aspects of the disease vary greatly with the superficial extent and the degree of the atrophous change. That fatty metamorphosis may be found, and to no insignificant amount, where neither subjective nor objective cardiac symptoms had awakened attention during life, is indubitable. Nay, it is equally certain that hearts have been carefully examined during life, and pronounced free from disease, which almost immediately afterwards (death arising from other causes) have been found very sensibly fatty. The disease may then to the observer of the present day, at least in its minor and moderate degrees, be *latent*. But the rule is that, if the affection be really well marked in amount, its clinical evidences are more or less precise and positive.

555. *Physical signs.*—The physical signs are, generically, those of a soft heart, free from notable enlargement.

(*a*) The impulse, always weak, is in extreme cases wholly imperceptible,—the precise point of the apex-beat, though ill-defined, can be caught better, both with the eye and finger, than in cases of attenuated dilatation with flaccid fibre. The existing indistinctness of the apex-beat is probably owing to the want of power of the fibres, naturally giving the spiral movement to the heart in systole; the whole organ is simply, and as it were lazily, projected forwards.

(*b*) The cardiac percussion-dulness, both superficial and deep, remains unchanged in area, unless there be alteration of bulk from some independent cause.

(*c*) Auscultation discovers, as a general rule, a weak, but by no means always toneless, short and relatively high-pitched, first sound; a long first silence, and a feeble, but relatively distinct and accentuated, second sound. If the fatty disorganisation be, as it often is, in great excess in the left ventricle, the first sound may be of notably fuller tone at the right than the left apex, and the second sound louder and more strongly accentuated at the second left, than at the second right, cartilage: the relative state of preservation of the muscular structure of the two ventricles explains these facts. I have never met with absolute deficiency of either sound over the entire cardiac region; but it is not uncommon (quite independently of the presence of any apparent syncopal condition) to find the first sound at the left apex almost null from weakness.

Basic systolic murmur may arise from coexistent anæmia; valvular murmur of any type from special valvular disease: both kinds are rare. But may the disease out of its own conditions generate murmur? I have for some time had under observation a case in which the whole symptomatology points to the existence of moderate fatty disintegration, and in which sometimes at the periods of syncopal and pseudo-apoplectic seizures, sometimes at other periods, a most distinct though feeble systolic murmur becomes *temporarily* audible at the left apex. Is this murmur of dynamic reflux mechanism?

556. *Symptoms.*—Nothing can be more variable than the mode of grouping, or than the intensity of individual symptoms. Between the extremes of latency, on the one hand, and conditions of cardiac, encephalic, and pulmonary function so perverted, on the other, that the continuation of life seems an impossibility, all conceivable shades of degree are observed. We must assume in our general outline of the patient's state, that the characters of the disease are, if not excessively marked, at least well and distinctively defined.

(*a*) The patient whose heart is notably fatty, though not of necessity in a low state of general health, is unable to undertake sustained labour, being exhausted on the first attempt almost, and often forced, when making an effort to walk, to stop at every few paces from cardiac uneasiness or dyspnœa, or both. In temper he is dejected and peevish,—emotional states coming either directly of the disease and its perverted state of circulation, or indirectly of the incapacity for occupation it entails. His tissues generally are soft and flabby; his muscular power almost null; the skin of the face pale and sallow, or more or less livid; the feet and ankles disposed to slight, but rarely, if ever, the seat of marked, œdema [512, *a*, *b*]; the appetite feeble, the digestive forces languid, the bowels confined, the liver larger than natural, and sometimes detruded downwards, sometimes distinctly increased in size, during paroxysms of dyspnœa.

(*b*) The state of the respiration varies widely in different cases, and at different times in the same case. The patient, though easily put out of breath, as a rule, and subject to fits of dyspnœa of very irregular rhythm and sometimes orthopnœal, yet not constantly short-breathed, may, on the contrary, be free, even under the pressure of great cardiac distress, from subjective or objective difficulty of breathing. So, too, may similar freedom of respiration hold on, on the eve, and during the occurrence, of the syncopal and apoplectic seizures incident to the disease.

A tendency to sigh, without emotional influence or mere habit to explain it, is in some cases connected with fatty disintegration. Again, in certain instances the breathing exhibits those peculiar characters, in regard of rhythm and intensity, first noticed, as far as I know, by Cheyne in 1816, and which are specially described further on [655].

Cough, of peculiar character, inasmuch as it is unattended with signs of bronchitis, or any other pulmonary affection, occasionally proves a troublesome symptom: it is, plainly, reflex and hæmic.

Seeing that pulmonary apoplexy is an occasional effect of flabby, or of hypertrophous, dilatation of the left ventricle (conditions tending to stagnation within the heart), it would follow that the weakness of fibre in fatty metamorphosis should conduce to blood-infiltration in the lung. Still, as matter of fact, the condition has not fallen under my notice, unless where dilatation coexisted.

(*c*) The pulse may be: (a) perfectly regular in force and rhythm, and of medium frequency, as the habitual state, even where strong evidence exists of advanced fatty change,—nay, in such cases even when the pulse becomes paroxysmally frequent—that is, relatively frequent—with anginal pain, it may maintain its regularity. But this is the exception. (b) Commonly the pulse is irregular in force and rhythm, either constantly or from time to time under the influence of excitement, flatulence, indigestion, or effort. On such occasions it may become exceedingly frequent, actually uncountable—in the main, from frequency; in part, however, from irregularity. The pulse may, when frequent and irregular, have also a tingling character, according to Stokes: but in the only instance he reports, there was marked mitral coarctation. (c) A third, and more noteworthy and special, condition of the pulse is infrequency,—sometimes carried to a very remarkable extreme, the beat falling, for instance, to twenty-six per minute.* Such infrequency is in some cases referrible to extra-weakness of occasional systoles, which fail to impress the radial wave; but, as originally noticed by Adams, the ventricular systoles themselves are occasionally much less frequent than natural: and, in point of fact, defect in the cardiac action proper is the really important condition. The infrequent beats may individually be either slow and lagging, or quick and abrupt.

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* J. B., private cases, May, 1854.

The mechanism of infrequent action has already been discussed, and the mystery, attached to it, admitted [285]. But it is yet more difficult to fix what are the conditions in different cases specially productive of the three main varieties of pulse just described. The greatest irregularity may exist without mitral reflux; but not, I am inclined to think, without dilatation.

Occasional fits of palpitation, attended with choking sensations, cardiac uneasiness, pain or actual angina, occur; and the patient, readily becoming faint on the least exertion, falls into a state of actual syncope from time to time.

Little or nothing is known positively concerning the state of the blood. I have failed to find the venous hum of anæmia, in cases where the tissues were markedly pallid,—but the patients were aged. The "piarhæmia," or oily impregnation of the fluid, noticed by Smith, was clearly exceptional and accidental.

(*d*) It is more than doubtful that the urine possesses any distinctive, or indeed habitually morbid, character: I can answer in the negative for fat, albumen,* sugar, biliphæin, excess of urea, casts of the tubules, and spermatozoa; oxalates, uric acid, and amorphous and triple phosphate, occasionally present, are of no significance,—except that they may point to the coexistence of an independent diathesis. As to the quantity of water habitually eliminated by this channel, without being able to furnish as precise information as might be wished, I can distinctly affirm that it does not oscillate in either direction notably out of proportion with the fluids consumed. This clinical truth seems somewhat at variance with the theory of eccentric pressure on the renal capillary system already referred to [491, *h*].

(*e*) Sexual inclination and power may both fail,—the latter more than the former. In one of the best marked cases of the disease, clinically speaking, I ever saw in a male under the age of forty-five, it was this failure, and not the cardiac symptoms, that led to my being consulted. Here, too, the genital organs were sound, and there existed neither spermatorrhœa nor spinal disease. Was the failure a mere coincidence, or really an effect of the languishing activity of the heart? On the other hand, I have seen many cases in which, as far as I could ascertain, there was no lack of erotic propensity or of copulative vigour.

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* Slight albuminuria occurs where dilatation coexists with fatty change; but I have not known the latter, unaided, produce it.

(*f*) The functions of the encephalon very habitually suffer more or less marked impairment. Little doubt can be entertained, that in many cases formerly set down as examples of pure dynamic disturbance of the brain, the groundwork of this lay in perverted circulation dependent on fatty metamorphosis of the heart.

As a general truth it may be fairly said that vertigo, failure of sight, dull aching and variously anomalous, indescribable sensations in the head, feebleness of intellect, and especially of memory, and somnolence, become common, with the advance of the disease, either as almost habitual states, or as paroxysmal occurrences.

The failure of memory is particularly striking,—like that attending chronic softening, it bears on recent rather than long-past *events*, and not on *words*: neither aphasia nor even amnesia occur, unless very exceptionally, in this form of heart-disease.

The psychical state, immediately preceding the syncopal attacks appertaining to the disease, is sometimes very curious. Thus a very intelligent patient, who has had a considerable number of such attacks, says: "for a minute or so before unconsciousness takes place, my memory of things just passed goes; I remember invariably, or rather my fancy conjures up, one of two or three different scenes that I have been witness of; there is no delusion, mental or optical, for I know they are not before me; but I think by shutting my eyes, I could realise them."

But these forms of disturbance may proceed much further. The patient, suddenly losing consciousness, may exhibit many of the appearances of an apoplectic seizure,—sudden fall to the ground, complete insensibility, with breathing either stertorous or of the peculiar rhythm to be by-and-by described [655], while the pulse becomes, even if previously an infrequent one, still more infrequent. The attacks are generally brief, easily recovered from, are very rarely followed by paralysis,—and in their immediate mechanism appear to me, as they have done to Stokes, rather to depend on deficient arterial supply and venous stasis, than on excess of blood transmitted to the brain. Stokes refers to a case in which the patient, warned by certain premonitory symptoms, was enabled to save himself from attacks by "hanging his head, so that it rested on the floor." There seems, indeed, a close alliance between these apoplectiform attacks and syncope; and a patient who has long been the subject of syncopal attacks, may lose these and have the apoplectiform variety instead. But, more than this, the seizure may be of a mixed apoplectic and convulsive character,—clonic spasms of certain parts coinciding or alternating with tonic spasms of others. Or the syncopal and apoplectic elements may be wholly deficient, the seizure being epileptiform in attributes,—marked by sudden fixed gaze, deathlike pallor of face, clonic spasms of the muscles of the head and neck, subsequently of the limbs, biting of the tongue, dilatation of the pupils, and on recovery total oblivion, or rather ignorance, of all that has passed during the paroxysm. I do not happen to have seen foaming at mouth, nor to have heard the epileptic cry, in attacks of this particular kind; but I have had the affirmative testimony of friends of patients as to the occasional existence of both.

Again, I have known a patient of this class, while engaged in ordinary occupation, sink on a chair, without actually falling, bend forwards as if to relieve suffering of some kind, become at the same time of deathly pallor, rapidly lose heat at the head and extremities, and utter no cry, while the pulse remains perceptible, no muscular twitch even occurs, and the pupils do not dilate. The consciousness goes completely in the objective, but apparently not wholly in the subjective, sense; waking up in a state of bewilderment, after a lapse of three or four minutes, the patient speaks of pain at the lower sternal region, of which pain *all memory disappears after a few minutes more.* Strange combination of cerebral and cardiac disturbances,—and which in more aspects than one seems to represent the state artificially induced by chloroform!

Again, I have lately seen a case in which temporary aphasia (at least in a subdued form) and amnesia occasionally occurred, unattended by any serious coincident disturbance of cerebration.

Now, the usual and perfect recovery after seizure in all these varieties would of itself suffice to show that they are unconnected with structural change in the brain. Neither is the converse form of proof wanting, for death has occasionally taken place after a repetition of them, and the cerebral substance been found free from any one of the admitted conditions of textural disease. Atheromatous and other degenerative changes in the capillaries are doubtless sometimes present, and may probably aid in promoting the blood-stagnation, on which the seizures in some cases depend. On the other hand, when, after apoplectiform seizure in a case of fatty heart, imperfect hemiplegia, quasi-idiocy, and mutism hold on (as I have known) for months, the reality of organic change cannot be questioned.

(*g*) The epileptiform character of the attacks would suffice in the opinion of some persons to prove implication of the spinal cord. In addition to this evidence it may be stated that in two cases, where abundant evidence of fatty metamorphosis existed, I observed formication, slight numbness, and tendency to subjective sense of cold in the lower extremities, the patients being free from other ordinary symptoms of disease of the cord, and certainly free from intra-pelvic affections, enlarged prostate, stricture of the urethra, renal disease, and spermatorrhœa. Were not these symptoms, indicative of implication of the medulla, analogous in mechanism to the quasi-apoplectic seizures?

(*h*) Thrombosis does not appear to occur often in the interior of fatty hearts, though the dynamic element of its occurrence, sluggishness of current, is so frequent in the affection.

557. *Course.*—As a general truth, fatty metamorphosis is a chronic process,—that is, though the immediate change, molecularly considered, may be, and probably is, rapidly effected, the extension of the change to any notable amount of structure, and its capability of producing appreciable symptoms, are the work commonly of months or of years. Nevertheless the fact, that individuals have occasionally been met with, noted for muscular prowess and feats of endurance almost to the day of their death, whose hearts notwithstanding have proved the seat of fatty conversion on a most extensive scale, seems to depose in favour of rapidly acute course as at least an occasional occurrence. And, as already stated, under the influence of adjacent inflammation, and in typhus, it seems tolerably certain fatty disintegration of the fibre may be acutely effected. On the other hand, it is conceivable that in the exceptional individuals just referred to (specially organised they must in any view be considered to have been), the supply of *vis nervosa* to the heart may be so great, as to render the contraction of a small mass of their heart-fibres as effective as the sum-total of those of ordinary persons. See, in the way of analogical illustration, the muscular work achieved by a feeble anæmic woman in hysterical paroxysms. But this important point in the history of fatty disease requires *ex professo* clinical study.

558. *Manner of death and prognosis.*—(*a*). Patients, whose hearts have undergone fatty metamorphosis, are often possessed with the idea, they shall expire suddenly. For this idea there is commonly a very obvious foundation, in the fact that they have

been more or less subject to various cardiac paræsthesiæ, or to actual syncopal attacks; but occasionally no such subjective hint has been, at least appreciably, given to the sufferer. In both classes of case the idea occasionally proves a prophetic one.

Instantaneous death may be of purely dynamic mechanism,—sudden syncope occurring from failure of the heart's intrinsic irritability, or from failure of motor innervation, or perhaps from excess of inhibitory innervation. Or such death may be mechanically caused by rupture and copious extravasation into the pericardial sac. Or, in cases of partial or minute rupture, death is at once statically and dynamically brought about. It has not occurred to me to witness or know of sudden death in those cases as a result of cardiac or arterial thrombosis. Rapid (but never actually instantaneous) death may follow apoplectiform and epileptiform seizures.

In many instances the fatal event is slowly brought about by asthenia.

(*b*) On the other hand, fatty disorganisation of the heart is by no means necessarily fatal. I have known *extensive* destruction of the kind exist, where death had occurred from unconnected chronic disease of other organs; and the proofs that a moderate amount of the change may be comparatively innocuous are of daily occurrence. May we go further, and accept the doctrine advocated by E. Crisp, that "fatty degeneration of the heart and arteries *generally*, in the aged, tends to prolong life by adapting the frequency and force of the heart to the requirements of the system"?* As already mentioned [494], it seems very reasonable to suppose, that weakening of the power of a *hypertrophous* heart through fatty metamorphosis may serve a useful purpose. But that the individual who, possessed in advanced life of a heart *normal in size for his age*, will prove a gainer, because its fibre partially degenerates into fat, seems a questionable piece of doctrine. If it were an advantage that the organ should be weak in advanced years, why does Nature steadily increase its mass, as time rolls on?

559. *Diagnosis.*—If weak cardiac action, a feeble toneless first sound, with a relatively well defined and accentuated second sound, absence of murmur, and percussion-dulness of normal area, coexist with frequent and irregular or infrequent and regular pulse, respiration of ascending and descending rhythm [655], and the peculiar cerebral attacks just described, there can pro-

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* Treatise on the Blood Vessels. Appendix, p. 363. 1851.

bably be little doubt of the existence of this form of atrophy of the heart. But the value of this proposition is less than it seems; for, even in cases anatomically well marked, the pulse may be perfectly natural; apoplectiform attacks are very rare; and the perverted rhythm of respiration equally so.

Besides, it is now established, as I suggested in the edition of 1854 would probably prove to be the fact, that other forms of cardiac disease, without a particle of fatty metamorphosis, may produce the special rhythm of breathing—and it is very probable, also, the peculiar apoplectiform seizures. In fine, the physical signs enumerated, coupled with deficiency of the dropsies and other systemic effects of dilatation, seem to furnish the most serviceable guides to direct diagnosis. Some help may occasionally be had from the notable perversion of the pulse-respiration ratio, entailed by the relative infrequency of the heart's contractions. Thus a pulse of forty may coexist with a respiration of twenty per minute,—a ratio of 2 : 1.

Canton has shown the arcus senilis to be the result of fatty atrophy of the cornea. But the arcus senilis, like fatty heart in the generality of cases, is an attribute of age: its discovery will do very little more in confirmation of an otherwise doubtful diagnosis of fatty heart, than would ascertaining that the patient has passed his fiftieth year. If indeed the corneal change were found in a youthful person, it might possibly have some weight: still, that fatty heart may exist without change in the cornea is certain, —and the converse holds equally good. The most remarkable specimen of fatty opacity of the cornea I ever saw in a person under forty-five years of age, so remarkable that the serious disfigurement attracted the attention of non-professional persons, occurred in a male cut off by chronic alcoholism, cirrhosis of the liver and ascites, who never exhibited the least clinical evidence of fatty disease in the heart.*

The direct diagnosis of the disease is consequently in an unsatisfactory state enough; nor is the clinical difficulty of the case always removed by contrast of the conditions of this affection with those of other diseases.

(*a*) Thus, positively distinctive signs of *simple flaccid softness* and fattiness of texture are in very truth wanting; and the chief safeguard against error lies in the extreme rarity of genuine cases of pure softening.

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* Mr. H., seen with Donald Fraser.

(*b*) Nor do I know of any means whereby fatty pseudo-metamorphosis may with surety be distinguished from *mere fatty infiltration.* The researches of Bizot into the relationship of subcutaneous fat to fat-accumulations about the heart furnish no clinically available help.

(*c*) The natural areæ of the cardiac dulnesses, and the absence of systemic congestion and dropsy, will distinguish fatty disorganisation from *dilatation;* if there be marked jugular distension, or *à fortiori* jugular pulsation, the case is not one of mere fatty disintegration.

(*d*) From all forms of *valvular disease* the present affection differs in the absence of persistent organic intra-cardiac murmur. But, as we have already seen, there is fair motive for believing that mitral regurgitant murmur of dynamic mechanism may temporarily occur. Basic systolic murmur of blood-origin is very rare; whatever be the morbid state of the fluid in these patients, it does not promote the occurrence of murmur.

(*e*) From *chronic pericardial effusion* fatty disease will differ by the normal area and shape of cardiac dulness, by the absence of præcordial arching, and by the fact that the heart-sounds are at least as well marked at the cardiac region as at the top of the sternum: the history of the case will, further, prevent the possibility of error.

(*f*) Supposing all commemorative story deficient, it may prove exceedingly difficult to distinguish one of the peculiar *apoplectiform attacks* intercurrent to this disease, at least at the actual moment of seizure, from uræmic coma. The breathing may, however, be genuinely stertorous in the former, while it never is so in the latter variety. In the pallor of face and absence of primary or sequential paralysis the two varieties agree. The condition of the urine, and the absence or presence of anasarca, will be the most available, but not infallible, guides to the diagnosis.

(*g*) Seaton Reid suggests that notable slackening of the pulse, when the peculiar respiratory disturbance, described further on [655], reaches its period of greatest effort or "distress," with increased frequency of the pulse during the period of apnœa, is a combination probably distinctive of fatty disorganisation.

560. *Treatment.*—The treatment of this affection is essentially that recommended for simple softening of the heart. It seems natural to enjoin abstinence from fats of all kinds, and of materials amylaceous and saccharine, readily converted into fat; yet there

may be little philosophy here after all,—if the local fatty state be, as seems strongly probable, a mere evolution of a form of atrophy. On the other hand, as to the propriety of a full diet of protinaceous food of easily digestible quality there can be no doubt. The vigour of the circulation requires sustainment from the moderate use of wine or spirits; and when, as is often the case, sufferers from the complaint have been habitual free livers, it certainly appears to me unwise (no matter what a tentative chemistry of the disease may suggest concerning the influence of an alcoholised regimen) to lessen their wonted daily amount of stimulus.

It is almost unnecessary at the present day to denounce the idea of letting blood in any form or degree for the disease itself, or for its accidental secondary phenomena.* Not only this, but the sufferer from fatty heart should be very cautiously submitted to lowering regimen or depressing medicinal treatment for any intercurrent attack that might, in a previously healthy person, call emphatically for such measures. The inhalation of chloroform is very certainly hazardous, though it is impossible to avoid the conviction that people with fatty hearts have occasionally inhaled with perfect impunity; nor can chloral be taken, unless in very small doses, without a certain amount of hazard. Chloroform liniments may be used to the præcordial surface, provided due caution be observed; they sometimes relieve cardiac distress very notably.

Of medicinal agents, iron, quinine, and strychnia are the most valuable. From the latter I have, as I believe, seen distinct improvement occur in the force of the heart's contraction. Digitalis seems occasionally useful,—and just in proportion as the pulse is frequent, and the physical signs point to the coexistence of dilatation; but, on the whole, the drug is not to be trusted in cases of this class. On somewhat of the same principle the gentle appliance of electro-galvanism seems seriously worthy of trial; but the necessity of caution in its use flows from statements already made (p. 193).

Moderate exercise on level ground, if not productive of distress, should be daily taken. Pure bracing air is theoretically indicated. But I am unacquainted with any facts proving that forced exercise in mountainous countries can cure, or even stay the onward progress of, the disease we have been considering; the cardiac affections I have known removed by Alpine tours, have been simply functional derangements of the heart dependent on gastro-hepatic dyspepsia.

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* Cheyne systematically bled in cases of fatty heart. Will it be affirmed that the "type of the disease" has changed since his day?

The apoplectiform seizures, intercurrent to the disease, should clearly be met by stimulants internally and counter-irritation to the lower extremities: they ought in fact to be treated much as syncopal attacks. Epileptiform paroxysms are plainly to be managed on ordinary principles.

I cannot close this brief account of fattily metamorphosed heart, without mentioning my strong persuasion that I have seen more than one instance in which, through hygienic and medicinal measures, such as those just mentioned, not only has the progress of decay been arrested, but real improvement actually effected in the nutrition of the heart. True, I should be puzzled to give positive demonstration that changes so notable had certainly been achieved; such demonstration is obviously unattainable. I found my belief on the facts that in certain cases where the physical signs, the local annoyances, and the general dyscrasia pointed strongly to fatty disorganisation, and to no other disease, as the cause of all, those signs have improved, those annoyances have disappeared, and that dyscrasia has given place to a diathetic state of relative tone and plasticity.

C, D.—AMYLOID AND SACCHARINE PRECIPITATES.

561. Amyloid degeneration has been seen in the heart and pericardium.* The symptoms, where the heart is concerned, will doubtless be those of ordinary fatty change,—but experience is limited.

562. Sugar has not, so far as I know, been found in the substance of the heart. But Bernard some years since detected diabetic sugar in pericardial serosity; the fluid, alkaline at first, became acid through the decomposition of the sugar.†

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* Friedreich in B. & F. Med. Chir. Rev., Oct. 1860.

† Bernard generalises this observation by suggesting that the excess of acidity in diabetic people, commonly regarded as an element in the causation of the disease, is really a mere effect of decomposition of already formed sugar.

CLASS II.—BLASTEMAL FORMATIONS.

SUB-CLASS I. DEPOSITS.

A.—SYPHILITIC DEPOSIT OR SYPHILOMA.

563. Constitutional syphilis may affect the heart, as it does various other internal organs: the fact seems to have been first distinctly proved by Ricord.*

564. *Form.*—Although there is evidence in some cases of interstitial infiltration of the connective tissue and interspaces of the muscular fibres [591], the syphilomatous mass, generally speaking, pushes these aside as it grows,—and the result is a clearly defined nodular product, varying in size from a pea to a nut.

565. *Anatomical constitution.*—As in the case of the lungs,† there is nothing in the anatomy of these formations to distinguish them from non-diathetic fibroid: their syphilitic nature is to be inferred from the antecedents of the individual, and the presence of indisputable evidences of external constitutional syphilis. This statement applies equally to naked-eye and microscopical appearances.

Cardiac syphilomata have been commonly found in the firm state, almost creaking under the scalpel; the case by Ricord shows softening may occur within their area. They have been seen of whitish-grey, pale flesh, or yellowish colour.

566. *Site.*—The walls of both ventricles seem to be their sole seat of origin,—the auricles escaping. Syphilitic infiltration probably occurs in, and thickens the substance of, the valves,—but I am as yet without anatomical proof of the fact: such clinical inklings on the subject, as I possess, will be found with the history of Valvular Diseases.

567. Syphilitic hearts have been found hypertrophous; but in one well recorded case (that of a female), the nutrition of the organ had failed, its entire mass weighing only five ounces.‡ The organ has commonly been free from valvular or other structural diseases.

568. Nodes, gummata in the limbs, syphilomata of the testicle,

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* Clinique Iconographique, &c., 1851.
† Diseases of Lungs, 4th ed., p. 513.
‡ John Morgan, Medical Press, November, 1868.

liver, and lungs, may give evidence of the true nature of the affection.

The internal organs may be free from disease of other descriptions,—the patient perishing of the syphilitic dyscrasia.

569. *Causes.*—The affection has been seen in both sexes.

Cardiac syphiloma may, like other constitutional evidences of syphilitic taint, be long in developing itself: in Morgan's case upwards of twenty years had elapsed since the first inoculation, before well marked signs of general infection developed themselves.

570. *Physical signs.*—Nothing definite is known concerning special physical signs. The results of percussion and auscultation have been the expression of the state of the heart's mass, and told nothing concerning the presence of nodules in its walls.

571. *Symptoms.*—Palpitation and præcordial uneasiness have been noticed, and, apparently on sufficient grounds, referred to the influence of the local accumulations.

572. *Mode of death.*—These patients have either died by the slow exhaustion of syphilitic poisoning,—when, of course, the manner of dissolution has had nothing distinctive about it; or the influence of the heart has been shown by the suddenness of decease. In the latter class of cases intra-cardiac thrombosis has occasionally been observed.

573. *Treatment.*—There is no evidence to prove (however probable the affirmative may be) that anti-syphilitic treatment reaches growths in the heart.

B.—TUBERCLE.

I.—CARDIAC.

574. Tubercle very rarely forms in the heart; and only, as a rule, in very small quantity and in cases of general tuberculisation, or as an extension from similar substance deposited under the pericardium. Its signs and symptoms, if it have any peculiar to itself, are unknown.

A case, remarkable for its exceptionality in almost all points, was observed many years ago by Townsend of Dublin. Here, while the lungs of the patient (a male aged sixty-one) contained only a few miliary tubercles, a tuberculous mass of some size existed in the walls of the heart, producing eventually fatal pressure on the pulmonary veins just at their line of union with the left auricle. Dyspnœa was the essential symptom.

II.—ENDOCARDIAL.

575. Minute tuberculous masses in the crude state, and also grey granulations, form under the endocardium in infinitely rare cases of general tuberculisation in the child. I know nothing of such deposits acting as the cause of endocarditis; though, on the analogy of the pericardium, it is very probable they may so act.

III.—PERICARDIAL WITH SEQUENTIAL PERICARDITIS.

576. But tubercle is a notably more common, though still a rare, formation beneath the serous layer of the pericardium, either cardiac or parietal. Occurring in the state of semi-transparent grey granulation, it may remain latent, or excite inflammation of that membrane. The ensuing pericarditis produces the same physical signs and local disturbance as if it were of idiopathic origin. But, probably on account of the tendency to secondary formation of tubercle amid the simple inflammatory exudation-matter,* the disease, unless fatal at once, tends, just as peritonitis of the same origin, to lapse into a chronic state, attended with more or less constant local symptoms. This was the view of Laennec; and it is supported by the course of a case that fell under my own notice.

577. In ordinary phthisis, as appears from the records of Louis, tuberculous pericarditis is very rare: it is vastly more uncommon than pleuritis or peritonitis, and notably so even than meningitis, of that form. It must not be forgotten that a phthisical patient may be seized with pericarditis, which shall be in no wise distinguishable in its products from that occurring in ordinary rheumatism, and which may prove rapidly fatal in the acute stage.†

578. *Diagnosis.*—Probably enough miliary tubercles, raising the pericardial surface, might by their abrupt collision generate a form of friction-murmur; but were such murmur heard, I know of no means by which the special nature of its mechanism could be determined. In point of fact, in the existing state of knowledge, its discovery would tend rather to mislead than enlighten the observer,—as it would obviously simulate the ordinary sign of acute exudative inflammation or, at least, congestion, with prominent vascular tufts [375], of the pericardium.

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* Carswell's Drawings, U. C. Museum, A. 533, fig. 2.

† Ryan, U. C. H., Females, vol. xiii., p. 43,—the solitary example of the fact, as far as I can remember, I have met with.

And even this sign, of deceptive import, will commonly be wanting; tuberculous deposit will of itself give no physical evidence of its presence, and the diagnosis of the disease can only be made through the pericarditis, it occasionally entails. But would the tuberculous origin of this pericarditis be announced by any peculiarity of its signs? Very certainly not; still, an attempt at divining its character might be made in the following way. If there were neither rheumatism nor "Bright's disease," nor adjoining pleuro-pneumonia present, and the patient had received no injury to the chest, and was distinctly of strumous constitution, the probabilities would be much in favour of the tuberculous origin of the inflammation. And this, too, although there were no distinct pulmonary signs of advancing phthisis; for, herein resembling tuberculous peritonitis, this form of disease has mainly been observed in cases where the lungs were but slightly affected.

But my recent experience has shown me yet another form of difficulty attending this diagnosis. A patient may have pericarditis of the most obviously scrofulous character, and yet not the slightest friction-murmur exist,—the absence of the sign resulting, as shown after death, from the excessive, almost greasy, smoothness of the strumous deposit.*

579. *Treatment.*—Although not so important a matter as in the days when profuse blood-letting was habitually recommended in acute affections, still even now the determination of the nature of tuberculous pericarditis is not a mere matter of diagnostic curiosity. The most appropriate treatment, as Burrows has successfully argued (Med. Chir. Trans., vol. xxx.), can scarcely be that adapted to rheumatic pericarditis. Blood-letting, if employed at all, should be had recourse to locally only, and in extremest moderation; mercurials likewise must be given with all needful care to avoid ptyalism: for my own part I would religiously avoid one and the other. Blisters in the neighbourhood of the præcordial region, dry-cupping, moderate purgation, diuretics with iodide of potassium and alkalies, are the agents on which, in connection with antiphlogistic regimen, reliance must be placed. Animal food may be allowed earlier in this than in other varieties of the disease.

SUB-CLASS II.—GROWTHS.

580. The heart is far from ranking as a frequent seat of growths. Primary production of any one of the series within the actual

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* Mercer, U. C. H., Females, vol. xiii., p. 7.

muscular substance is, in truth, rare; and when we take into consideration the extensive relationship of the endocardial surface to the blood, circulating absorbed elements from primary formations elsewhere, it seems remarkable how rarely the cardiac structures become the seat even of secondary development of the kind.

Syphilomatous fibroid, just described [563], enchondroma, fibro-ossiform tumours and cancer, are the only species of growth that have been observed in the textures of the heart.

ENCHONDROMA.

581. A woman, aged forty, six weeks before death, began for the first time to suffer from symptoms traceable to cardiac dropsy. On examination, several rounded, smooth, and slightly lobulated tumours, varying in size from a pin's head to a lump, an inch and a half in diameter, were found "over the heart and chiefly around the large vessels;" some of them lay on the parietal pericardium. True hyaline cartilage existed in islets within fibro-cartilaginous substance: there was no bone seen, but calcareous salts had been deposited. There were thirty ounces of fluid in the pericardium.*

The absence of symptoms until within six weeks of death accords with the results of observation of other kinds of cardiac tumour. Was the pericardial fluid dropsical, and the effect of pressure of the growths on the veins? [459, *d*].

FIBRO-OSSIFORM GROWTHS.

581*. Some specimens of the kind exist in museums, but without clinical history.

CARCINOMA.

I.—MUSCULAR SUBSTANCE.

582. Cancer of the heart is by no means so extraordinarily rare, as is commonly supposed,—that is, if secondary and clinically insignificant, as well as primary and grave, examples of the disease be included.

Cardiac cancer may be the solitary evidence of the diathesis, or it may occur in combination with similar disease elsewhere.

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* Kelly, Brit. Med. Journal, 1872.

When thus associated, which is vastly the more common state, it is, as far as I know, always secondary.

583. *Anatomical characters.*—Encephaloid is essentially the species found in the heart; examples of genuine scirrhus are very rare; while of colloid I have neither seen nor read of a well-defined instance. Melanic pigment is sometimes more or less copiously deposited in the stroma and within the loculi of encephaloid masses,—a deposition not impressing in this locality, more than elsewhere, any special character on the cancerous substance.

The nodular is the usual form of deposition; but the sarcous substance may be infiltrated, like the muscles of the limbs, in such manner that portions of the heart seem to the eye metamorphosed into cancer. And the actual atrophy of sarcous structure is, under these circumstances, very extensive,—but evidently not complete, as a case to be by-and-by narrated would prove, as well as a yet more remarkable one in which eleven-twelfths of the organ are said to have been involved. Had the conversion been here as complete, as it seemed, life evidently could not have held on.

Nodules may form under the endocardium and pericardium without implicating the heart's fibres,—a fact of which I have elsewhere published a striking example.* Or they may similarly grow within the muscular substance,—and in vast numbers, as the case recorded by Dupuytren, in which he ceased to count at the six hundredth, sufficiently proves.

The part of the organ involved varies greatly; in fact every conceivable combination of localities is exemplified by actually observed cases. It does not clearly appear that the right compartments exhibit any notable excess of proclivity to the disease.

When the disease was nodular and secondary, I have found the heart's texture, otherwise, in all respects perfectly natural; nay, even in cases of infiltration, the fibre just beyond the implicated islet may be thoroughly sound. There is no disposition to myocarditis, local or general; but recent endocarditis in the immediate vicinity of the cancerous formation has occasionally been found, as well as old pericarditis. The presence of dilatation or hypertrophy is purely accidental.

584. Cancer of the heart does not spread to other organs, but conversely extension of the disease from the mediastinum and

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* Clinical Lecture on Subcutaneous Cancers, Medical Times, August, 1852.

lung to the heart is not very uncommon, where primary accumulation has occurred in those localities. Some particulars of a remarkable case of the kind are detailed a little further on. And this appears to me to be the only manner of localisation of primary cancer in which the heart exhibits any particular tendency to suffer secondarily. Nevertheless, parts the most distant, the most diversified, and the most functionally disconnected with the heart, being the primary seats of the disease, the latter organ has now and then become secondarily involved,—while, singularly enough, the liver and lungs, the usual seats of secondary growth, had escaped contamination.

585. *Causes.*—Males are very notably more prone to suffer from cancer of the heart than females; of twenty-eight cases, including primary and secondary formation, three only were furnished by females. The disease occurs at all ages; if a narrative of Billard may be accepted literally, scirrhus occasionally forms in the heart even during intra-uterine life.

586. *Symptoms.*—Cancer of the heart has in the majority of recorded instances remained wholly latent. Such absence of symptoms is not to be wondered at, where minute or even pea-like nodules only have formed in the substance of the organ; but, where pretty extensive infiltration of the walls had occurred, the deficiency of both signs and symptoms affords fair matter for surprise.

Still such deficiency is not invariable, as the following particulars, condensed from a report by Andral, show. A man, aged thirty-seven at death, began, two years previously to that event, to suffer from slight dyspnœa. This was followed five months later by a sudden and temporary attack of violent præcordial pain, severe dyspnœa, vomiting and loss of consciousness; the succeeding year the habitual dyspnœa increased, and seven or eight paroxysmal attacks, such as just described, occurred; six weeks before death emaciation and straw-coloured tint of skin were observed. In the intervals of ease nothing could be detected by auscultation. Anasarca, commencing with the lower extremities, reached the upper limbs and the face; and the patient died suddenly without struggle. Here combined cancerous infiltration and tumour had almost completely taken the place of the muscular structure of the right heart. No other morbid change, it is affirmed, could be detected in that or in any other organ.*

But there can be no surety that these symptoms were not rather the produce of obstruction of the large veins than of the change in the heart's structure. I am certain, grave infiltration may exist without impressing any specific character on the symptomatology. Thus, in a case recently seen,* where the physical signs and a variety of "concentric pressure-symptoms"† enabled us to diagnose the existence of mediastinal tumour and left pleural effusion, the following are the notes of the examination of the heart:—"Slight cardiac impulse at left costal angle, very faint in proper heart-region; no cardiac impulse or thrill to the right of the sternum or above the clavicles. . . . Heart's sounds remarkable alone for their pendulum-like rhythm; no murmur at left apex; nothing abnormal in sounds in the course of the arch of the aorta; pulse 120, excessively feeble, regular, a little fuller in left than right radial. . . . Close to the inferior angle of the left scapula is heard a strongly marked, rough, medium-pitched arterial murmur." There was no clinical evidence, then, pointing to the heart; and yet the anterior portion of the wall of the right auricle was so infiltrated with cancer, as to seem metamorphosed into that substance, while the mediastinal mass pressed seriously in the direction of the limited space between the tricuspid and pulmonary orifices. The capacity of the right ventricle appears to have been reduced by the pressure of the encroaching growth. Both the left cavities were of natural extent, and the mitral valve and orifice normal. The dorsal murmur in the descending aorta was doubtless caused by direct pressure of some portion of the tumour. Immediately beyond the infiltrated substance, the muscular texture was perfectly natural.

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* Anat. Pathol. t. ii., p. 328. 1829.

587. The diagnosis of cardiac cancer has never, as far as I am aware, been made.

II.—ENDOCARDIUM.

588. An example of presumed cancerous ulceration of the endocardium has been recorded by Payan.‡ The details given appear anything but conclusive as to the genuinely cancerous nature of the ulcerations described. Nor is there any proof that the encephaloid mass found by Hudellet attached to the wall of the right auricle, almost filling its cavity, and extending into the ventricle and both venæ cavæ, was really produced from the

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* Scammell, U. C. H., Males, vol. xvii., p. 275; and Univ. Coll. Museum, No. 4580.

† Diseases of Lungs, 4th ed., p. 167.

‡ Revue Médicale, Nov., 1841.

endocardial surface. Probably cancer-elements underwent evolution in an enlarging blood-coagulum. The primary disease lay in the parotid glands. No chest symptom, singular to relate, had existed; death occurred instantaneously after a meal.*

III.—PERICARDIUM.

589. Minute cancerous nodules occasionally form under the serous membrane, both parietal and cardiac. But, even if they acquire some size, they will not only fail to produce symptoms, but will cause no friction-murmur, unless the serous surfaces be rough.†

A case of accumulation of cancerous substance in layers within the serous sac, recorded by Bright, ‡ is rendered peculiarly interesting by the occurrence of aggravated attacks of paroxysmal dyspnœa, mainly taking place by night, and accompanied with general agitation and spasm of the diaphragm. These seizures were probably direct and reflex results of irritation of the phrenic nerve embedded in the morbid mass.

SUB-CLASS III.—PSEUDO-TISSUES.

INDURATION-MATTER.

CIRRHOSIS.

590. Infiltration of the connective tissue and muscular interspaces of the heart with exudation, which hardens into induration-matter of low plastic and contractile type, is a very real, though a rare, morbid condition of the organ.

Assimilable in nature to cirrhotic infiltration in the liver, kidney and lung, in the heart also the induration-matter is laid down, in sequence to congestion, and not to an actively inflammatory process: the disease is not chronic myocarditis. But, whereas the former organs are very commonly affected on an extensive scale, if affected at all, limited portions only of the cardiac substance undergo the change,—either an islet of the ventricular walls, or some portion of the columnæ carneæ, especially the papillary muscles.

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* Cruveilhier, Anat. Pathol., Livraison xxix.

† Clin. Lect. on Multiple Subcutaneous Cancers, Med. Times, 1852.

‡ Med. Chir. Trans., 1839.

The infiltrated material is sometimes structureless, sometimes obscurely fibroid. It may undergo fatty metamorphosis, or become calcified, or even pseudo-ossified. I have seen the latter condition of a papillary muscle attended with the like alteration in some of the attached tendinous cords. Atrophy of the proper muscular texture proceeds *pari passu.*

The inherent disposition of induration-matter to contract is, in the wall of the ventricle, controlled and overcome by the eccentric pressure of the blood; and the resistant power of the material being small, the result is local distension. True sacculated aneurism of the heart originates more frequently in this than in any other manner. Rupture of the dilated part of the wall of the heart may eventually occur.

591. Although commonly traceable to passive, sometimes to mechanical, congestion, this cirrhotic state of the heart's texture occasionally occurs independently of any perverted state of local circulation. At least no sort of evidence can be found in some cases, either in the past history or the *post-mortem* appearances, warranting a belief in such perverted state.

Just as in the case of the lungs, cardiac cirrhotic infiltration is occasionally observed in the subjects of chronic alcoholism and of tertiary syphilis [564].

592. This alteration is excessively rare in the walls of the ventricles, and almost confined to the left; but a greater or less degree of the change is not very uncommon in the papillary muscles. Now in the former situation it is not revealed by either symptoms or signs, unless it have proceeded to such lengths as to have induced aneurismal pouching, of which the clinical effects, elsewhere described, will prove more or less obvious. In the latter situation the infiltration must of necessity, if it have proceeded far enough to cause contraction of the papillary muscles, entail imperfect closure of the mitral orifice with the murmur distinctive of that state.

CLASS III.—PARASITES.

ENTOZOA.

593. The heart occasionally affords a nidus to cysticerci, and to acephalocysts. I am not aware that the trichina-spiralis has ever been found in its substance.

ACEPHALOCYSTS.

594. It would appear that multiple broods are rare in the acephalocysts of the heart,—generally, indeed, the mother-cyst is solitary. In this organ, as elsewhere, the cysts afford a habitat to the echinococcus.

595. Sometimes acephalocysts have coexisted in their common seat, the liver; occasionally the heart alone has been affected.

596. The septum of the ventricles appears to be a favourite cardiac seat of these entozoa.

597. *Symptoms.*—Symptoms do not occur as matter of necessity: their absence or presence will depend on the mechanical influences exercised by the sac.

(*a*) Thus among Carswell's unpublished drawings appears the figure of a heart containing in the posterior part of the wall of the left ventricle a good-sized acephalocystic sac, protruding somewhat on the pericardial surface.* The patient, a female, was cut off by phthisis, and the sac accidentally found; and as she had died in hospital, and the heart had not attracted attention, the probability is there were no cardiac symptoms.

(*b*) On the other hand sudden death may be caused by the mechanical influence of these productions. Thus I have before me a heart containing an acephalocystic sac, as large as a pigeon's egg, in the septum of the ventricles:† the patient, a female, dropped dead suddenly, while engaged in her household affairs. Rokitansky relates a case in which an acephalocyst, nearly as large as a hen's egg, escaped from the mother sac in the upper part of the ventricular septum, and caused instant death by becoming impacted in the conus arteriosus and pulmonary artery,—reaching almost to the left branch of this vessel. Kelly‡ records the sudden death of a boy, aged ten, from rupture of a similar cyst into the right auricle: each main branch of the pulmonary artery contained a sub-cyst.

§ VIII.—DISEASES OF THE ORIFICES AND APPENDAGES.

598. Diseases of the orifices of the heart, though not wholly unproductive of pain and other forms of subjective inconvenience,

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* Univ. Coll. Mus., A. 9.

† Univ. Coll. Mus., No. 2293.

‡ New Syd. Soc., 1871, p. 158.

are essentially injurious through their mechanical effects. They interfere in either, or both, of two ways with the circulation through the organ. They are constrictive or stenotic in anatomical character, and hence produce *obstruction* of the onward current: or they entail disproportion between the size of the orifice to be closed and the valve to close it: or they interfere with the play of the closing apparatus, and in either of these two latter fashions lead to *reflux* or *regurgitation* of a backward current. In all three cases impediment to the onward-moving stream is the result.

599. Each orifice is in theory capable of each kind of disease, —obstructive and regurgitant; but the frequency, with which the various orifices actually suffer, differs very widely. This question, however, requires to be considered in two points of view, the anatomical and the clinical: the excess of frequency of organic change in the actual texture of the valves and orifices of the left side is very great; the excess of frequency of disturbance, clinically demonstrable, in the functional conditions of the orifices of the left side, much less. This comes of the fact that the tricuspid valve is very often incompetent through mere widening, without textural change, of the orifice, while its own structure remains sound: the excess-cases of textural disease of the valves on the left side are counterbalanced by the excess-cases of misfitting of one of the closing valves on the right.

600. Obstruction with stenosis of orifices, *as far as is known positively*, is always of organic character, and is caused by morbid change in the valves, in the wall of the orifice itself, or by extraneous pressure. Regurgitation through orifices is, certainly, in the vast majority of cases, likewise of statical mechanism, and is caused by morbid change in the valve, in the wall of the orifice, or in some part of the apparatus connected with the closure of the valves. But it may be held to be certain that regurgitation may be produced dynamically in the mitral orifice at least, through functional imperfection of the closing apparatus, though this be perfectly free from organic change: regurgitation of this kind, is, however, of temporary duration, and does not produce results of importance. It is possible, too, that obstructive disturbance may be produced at the mitral orifice by perverted action of the papillary muscles; and either regurgitation or obstruction at the mouth of the aorta by perverted innervation of the sygmoid valves: but all these three latter propositions are completely hypothetical at the present day; and hence, practically, the serious disturbances at orifices may be regarded as organic and statical.

601. Diseased valves, diseased orifices, and diseased states of the apparatus effecting the closure of the valves, are, then, the agents of perverted circulation through the outlets, and through the channels of communication between the cavities, of the heart.

(*a*) *Diseased valves* are the causes of *obstruction:* first, through local endocarditis and its products interstitial and superficial, alterations of form, and adhesions of the divisions, of the valves *inter se;* secondly, through deposits of fibrine from the blood on their surfaces; thirdly, through atheroma, calcification and their sequences; and, fourthly, in some instance, through hypertrophy of the valvular tissues.

Diseased valves, again, are the source of *regurgitation:* first, through endocarditis and its effects (especially when these tend to produce puckering and diminished superficial size, or actual destruction of the cusps of the valves, or adhesion of these either to each other, or to the adjacent wall of the heart or great vessel to which they belong, or to entail thickening and shortening of the chordæ tendineæ, or rupture of these); secondly, through fibrine deposited among, and interfering with, the action of the chordæ tendineæ; thirdly, through atheroma and its sequences; fourthly, through hypertrophous thickening of the valves interfering with the freedom and completeness of their fall at the proper moment; fifthly, through atrophy of the valves, lessening their depth or superficial extent, in rare instances affecting their continuity by extensive perforation; and, sixthly, through atrophous shortening of the chordæ tendinæ.

Besides, certain conditions may exist at an orifice causing at one moment obstruction, at another reflux, of the current [119, *b*].

(*b*) *Diseased orifices*, the actual substance of the valves being sound, lead to *stenotic obstruction* through the products of endocarditis, atheroma, and calcification lying upon, or producing contractile and other changes in, their surfaces or walls. And disease of the orifices is the cause of *regurgitation*, where, the valve being sound, the opening is morbidly widened to such an extent as to render the former inadequate to its closure.

(*c*) *Diseased states of the apparatus*, connected with the closure of the valves, are seen in induration and cirrhosis with diminished bulk of the papillary muscles, and in shortening of the chordæ tendineæ; they produce *regurgitation*. I once met with a direct mitral murmur, where the orifice, instead of being obstructed, was actually too wide; a chorda tendinea from the long tongue of the valve bifurcated, each division attaching itself to a separate papillary muscle, and so placing a thread-like obstruction in the way of the current.* It may be made a question, too, whether imperfect action of the papillary muscles, from textural disease of their substance, may not act obstructively also, and throw an obstacle in the way of the passage of the blood from the auricle to the ventricle.

602. Such are the general characters and immediate effects of the morbid state of the orifices and their connected apparatus. We will now pass to the consideration of the signs and symptoms of these morbid states at each orifice. These signs and symptoms, it must be noticed, are essentially of mechanical origin; and hence their discovery discloses the existence simply of obstruction or regurgitation at a certain orifice, but tells nothing directly, and by necessary inference, as to the nature of the morbid process, whether inflammatory, fatty, calcifying, fibrillating, exudative, ulcerative, or cancerous, that has engendered the mechanical difficulty. In the majority of cases valvular disease entails some form of anatomical change in the substance of the heart itself; the proper signs of the former and of the latter are, therefore, met in frequent clinical association; for purposes of precision, it is necessary each class of signs should be enumerated apart,—as is done in the following descriptions. As already shown, too [117], different valvular affections often coexist, and in certain combinations the one may tend to throw into the shade, or actually modify, the signs of the other or others.

603. To save the necessity for repetition with the account of each valvular disease, it may be stated here, once for all, that *valvular disease never of its own self alters the area or intensity of the heart's dulnesses, superficial or deep-seated.* If either be affected, the presence of some other morbid state becomes matter of certainty.

MITRAL REGURGITATION, REFLUX, OR INSUFFICIENCY.

604. How is the reality of mitral regurgitation during life to be demonstrated after death? Tie the aorta and coronary arteries, cut off the apex of the heart, and let fall against the mitral valve a full-sized column of water: the valve will either support the column, or allow it to pass through to the auricle. If the whole of the ventricle above the floor formed by the valve remain filled with the water, scarcely a drop filtering through, there can be no

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* Pickett, U. C. H., Males, vol. vi., p. 231.

doubt the valve was competent during life.* If, on the other hand, the water escape rapidly, the incompetence of the valve may be held certain. But in the case of slight filtration through the orifice, it would scarcely be justifiable to infer the insufficiency of the valve during life, as we have no means of imitating the vital contractions that accompany the act of closure in the living and moving heart.

605. *Physical signs proper.*—The impulse, in highly marked disease of this species, is, in the majority of cases, irregular in force and rhythm. Systolic thrill may sometimes be felt at and about the left apex [63, *a*].

But the essentially distinctive sign of this regurgitation is systolic left-apex murmur,—possessing the various attributes already fully described [109].

It is shown in the description referred to, that several other conditions besides reflux of statical mechanism through the mitral orifice may produce murmurs of similar time and site [vide Table, p. 95]. And the difficulties standing in the way of sure diagnosis are there considered. Well-defined audibleness of the murmur in the vertebral groove is one of the most significant diagnostic characters.

The state of the cardiac sounds themselves is as follows. The systolic sound, completely or incompletely covered at the apex by the murmur, may be perfectly natural at the ensiform cartilage and at the mid-sternal base. The second sound, often weakened, in consequence of diminished calibre of the aorta, at the second right, or aortic, cartilage, is accentuated in many, but, as we have already seen [109], not in all, instances, at the second left, or pulmonary, cartilage. Sometimes the second sound is distinct and sharp at the left apex, much sharper than at the pulmonary cartilage; a fact illustrated by the case (Anne Gippin) quoted a little further on [609].

606. *Indirect Physical signs.*—Dilated hypertrophy of the left ventricle is the common sequence of mitral regurgitation,—and is habitually associated with thickening and enlargement of the same auricle. The direct physical signs of these conditions consequently play the part of indirect signs of mitral reflux. The hypertrophy itself is plainly of the supplemental species; though it is difficult to see how a condition, which increases the vehemence of recoil

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* Such was the fact in Wilkinson, U. C. H., Males, vol. ix., p. 306; Landers, U. C. H., Females, vol. ix., p. 289; Fosbury, U. C. H., Males, vol. x., p. 133; Doyle, U. C. H., Males, vol. x., p. 238; Snail, U. C. H., Females, vol. x., p. 260.

through the affected orifice, can tend to balance the evils arising from reflux.

(*a*) The apex-beat and impulse generally are carried outwards, and lowered somewhat. The impulse is increased in force, and in rare instances there is auricular impulse at the second interspace, —either pre-systolic, when it comes of hypertrophy of the auricle, or systolic, when it is communicated from the ventricle.

(*b*) The area of percussion-dulness is increased, especially to the left: but, as a result of sequential hypertrophy of the right ventricle (which I have oftenest observed in early youth), extension of dulness may eventually take place to the right also.

(*c*) The auscultation signs vary, of course, with the amount of the enlargement. If the auricular hypertrophy be well-marked, præ-systolic knocking sound may sometimes be caught about the third left cartilage [103, *k*, 2].

607. *State of the Pulse.*—The condition of the pulse in mitral reflux is not only variable, but its variations do not bear any constant relationship to the amount of anatomical defect at the orifice concerned. Four different types of pulse have fallen under my notice:

(*a*) A pulse fairly natural in all its characters,—in this case, however, reflux is always, as far as I know, slight in amount.

(*b*) A pulse regular in force and rhythm, small, short, occasionally sharp, rather frequent, and compressible, unless (and sometimes even in spite of this) marked hypertrophy coexist.

(*c*) A pulse irregular in force and rhythm, sometimes to an excessive degree, small, short, feeble, occasionally sharp, and tremulous or polycrotous under excitement of the heart.

(*d*) A pulse such as that last (*c*) described, alternating non-rhythmically with comparatively full, strong and prolonged beats.

In all the three last conditions the cardiac systoles and radial pulses may fail to correspond in number, in strength, and in normal relationship of rhythm [103, *i*].

There seem to be two kinds of reason, why no precise correspondence holds between the probable amount of reflux and that of disturbance in the pulse,—the one dynamic, the other statical. First, it is plain the heart suffers dynamically in its rhythm in some cases of mitral reflux, just as it may suffer from perverted innervation, where no mitral incapacity exists at all [279]. Secondly, as (originally, I believe) taught by Marey, if stenosis of the orifice be added to insufficiency of the valve, the pulse *may* become regular. Of this clinical fact (of which a fanciful explanation may easily be found,—the ventricle, receiving less blood, encounters less difficulty in disposing of it) I have met with some very satisfactory examples. Among these may be noted the case narrated in excerpt further on [609]; where, though the conditions of reflux were carried to the highest point, the pulse was regular in rhythm, marked mitral stenosis coexisting. I think that aortic stenosis also tends to regularize the pulse of mitral reflux.

The sphygmogram of the pulse varies just as its tactile characters; and the curves exhibit no peculiarity by which the observer may identify their unevenness as dependent on mitral regurgitant, rather than on purely neurotic, irregularity. (*Vide* APPENDIX.)

608. *Effects on the Capillary systems.* (*a*) *Systemic.*—Pure mitral regurgitation produces but little effect on the systemic capillary circulation. The disease may exist for years without inducing either general dropsy or distal venous stasis. If dilated hypertrophy supervene, systemic obstruction occurs with a facility proportional to the amount of dilatation and, especially, the impoverishment of the blood. The systemic conditions then become those of dilated hypertrophy in an aggravated form, *plus* certain others, to be by-and-by mentioned, more or less peculiar to the regurgitation. I use the word "aggravated" advisedly; it has not occurred to me to observe cases justifying the idea that mitral regurgitation acts as an efficient safety-valve to a dilated and hypertrophous left ventricle.

(*b*) *Cerebral.*—The disturbed conditions of the sensorial functions sometimes observed in mitral disease are likewise the results rather of the dilated hypertrophy with which it is associated, than of itself alone; still, when carried to a great amount, it may secondarily, through its influence on the pulmonary circulation and the right heart, tend to congest the brain passively.

Many years ago eight cases (four of them accompanied with *post-mortem* details) were adduced by Law, of Dublin, with the design of showing, that white, or colourless, softening of the brain, held to be identical in nature with gangrene of other textures, is frequently dependent on reflux through the mitral orifice. Law's observation was correct: there *is* an occasional nexus of cause and effect between mitral disease and imperfect brain-nutrition. But his notion that defective supply of blood to the brain, as part of the abiding failure of the systemic arterial current, acted as the cause of the cerebral necrosis, seems unfounded. At least in many cases (as first shown by Kirkes in 1852), the brain-changes arise from impaction of embolic fragments, detached from the valve, within the supplying arterioles. Fresh interest has been given to this matter by Hughlings Jackson's connection of aphasia (associated or not with amnesia and agraphia), through plugging of the left middle cerebral artery, with mitral disease.*

(*c*) *Pulmonary.*—The essential evils of mitral disease are pulmonary,—congestive and irritative. The current thrown back through the auricle on the pulmonary veins tends to congest the lungs; and it is not unreasonable to admit that the effort on the part of the right ventricle to overcome the increasing obstruction must induce serious irritation. Cough, watery expectoration, dyspnœa, and orthopnœa; actual bronchitis, pulmonary œdema, pneumonia, either congestive or irritative, mechanical congestion and pulmonary apoplexy, are the direct effects of the disease. The expectoration may, through the latter condition, be stained of dark blood-tint, or blackish; or actual escape of pure blood may occur: what maximum quantity of blood may be brought up from this cause at a time, I do not know; I have never seen any large amount.

(*d*) *Gastro-hepatic.*—The hepatic system gradually becomes engorged, as likewise that of the gastric mucous membrane, in sequence to the lungs and right heart. To this engorgement are due in great measure the various disturbances of digestion, observed in advanced cases of the disease,—and especially the obstinate nausea which occasionally renders yet more miserable the closing period of existence.

(*e*) *Renal.*—Traces, more or less considerable, of albumen may be sometimes found in dependence on abiding, or temporary, renal congestion. I think this is a very rare occurrence, however, unless the heart be notably dilated. I have not met with distinct renal embolism: but it does occasionally occur.

MITRAL CONSTRICTION, STENOSIS, OR OBSTRUCTION.

609. *Physical signs proper.*—If the left auricle be hypertrophous, and especially if the constricted orifice be rough, præ-systolic

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* Lond. Hosp. Reports, vol. i. On the whole subject of aphasia, as its natural history till lately stood, the reader may study with advantage the critical digest of Frederic Bateman (On Aphasia, &c., Lond., 1870). The original and luminous papers of C. Bastian (Brit. Med. Journal, May, 1869), and of W. H. Broadbent (Med. Chir. Trans., vol. 55, 1872) should be perused in sequence. It is not unworthy passing notice that the only firm grasp of the great problem in *physiology*—the mechanism of speech—has been obtained by meditation on facts in *pathology*. Therein appears one of the happiest conceivable illustrations of the fundamental truth, that while physiology may often suggest inferences in pathology, the latter branch of biology may play a yet more effective part in laying bare the secrets of the former.

thrill may be perceived [63, *a*]: such thrill is, however, often absent. The measure of the impulse varies, being either regular or irregular, in force and rhythm; in all probability the irregularity, when present, is in the main neurotic, even more so than in the case of reflux disease [607]: regularity of rhythm is the rule.

The special physical sign of mitral stenosis, præ-systolic left apex murmur, is described elsewhere [113]. This murmur is, however, as already stated, very frequently deficient. And it is important to note that, even where a hypertrophous auricle lies behind a constricted mitral orifice, no murmur *necessarily* ensues, if the narrowed surface be smooth, nay, even if it be rough,—this is proved by the following excerpt:—

"Anne Gippin, ætat. 35. . . . . p. = 144, regular in rhythm, irregular in force, feeble, small; r. = 48; anasarca; ascites; jugulars swollen, pulsatory; radials faintly visible at wrist; heart's action too extensive,—impulse slapping rather than heaving; faint thrill at apex-region. At mid-sternal base both sounds dull; first slightly murmur-like, and accompanied by knock; at the left apex systolic roughish murmur; while the second sound is distinct and much sharper than at the pulmonary cartilage. . . . . *Post-Mortem* (twenty days later): heart, with 1½ inches of great vessels, weighs 13½ oz.; tricuspid orifice measures 4⅞ inches; pulmonary orifice measures 3⅛ inches; the tricuspid valves looked insufficient to fill the widened orifice, but no experiment done; left auricle very hypertrophous, in some parts wall = ¼ inch in thickness, its endocardium creaks in cutting; mitral orifice just admits end of index-finger, measures 1⅞ inch; edge is rugose, and valve is directed in funnel shape towards the ventricle; cordæ tendineæ, very thick, are so shortened that practically the papillary muscles seem inserted directly into the valves. Aortic orifice,—water passes gradually through from above (but the coronary arteries were not tied); fine warty yellow vegetations, especially on corpora Arantii; width of orifice = 2⅜ inches." . . . . (U. C. H., Females, vol. v., p. 292-299, Dec. 1850.) See, also, case of Denham, note, p. 101.

This case, taken alone, would suffice to show what little faith is to be placed in Skoda's test for the distinction of præ-systolic murmur, caused by stenosis of the orifice, from that resulting from mere roughness of its endocardial surface; for here, although the constriction was very great, no accentuation of the second sound existed at the pulmonary cartilage.

The above case has been given special interest in consequence of its having been selected by Herbert Davies * to illustrate the manner in which the amount of abnormal change of area of certain orifices may be calculated on the assumption that certain others are healthy [34, *d*]. Assuming that the tricuspid opening was of about normal area (though it was in reality seven times larger than the morbidly contracted mitral orifice, instead of being only 1·3 to 1·4 times larger, as in health), then as—

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* Proceedings Royal Society, March 17, 1870.

As $\dfrac{\text{Area of tricuspid}}{\text{Area of mitral}} = 1{\cdot}3$ nearly

$\therefore$ Area of mitral (healthy). $= \dfrac{\text{Area of tricuspid}}{1{\cdot}3}$

$= \dfrac{1{\cdot}9}{1{\cdot}3} = 1{\cdot}45$ sq. in.

Hence the amount of contraction of the mitral orifice $= 1{\cdot}45$ the normal
$-\ {\cdot}28$ its actual size
$= 1{\cdot}17$ sq. in.

As to the condition of the heart's sounds themselves: it will be seen, that in the above case the *second* sound was clearly defined at the left *apex*, there being no constrictive murmur; but that sound will be audible there, even when mitral constrictive murmur actually exists. On the other hand, so frequent is the association of regurgitation, that it is rare to find a pure *first* sound at that point. At the mid-sternal base the first and second sounds are of natural character, except that they are sometimes feeble, from the smallness of the current sent on from the left ventricle; or occasionally (not, I think, with the constancy taught by Bouillaud) reduplicate on the principles already explained [103, *k*, 2]. At the aortic cartilage, *both* sounds are feeble; at the pulmonary, both, on the contrary, and especially the second, may be full and accentuated: if there be coexistent insufficiency of the tricuspid valve, this accentuation will, however, as in the case just referred to, be prevented from occurring. Again, both sounds may be natural and well defined at the "tricuspid apex" or ensiform cartilage.

610. *Indirect Physical signs.*—Left auricular præ-systolic impulse, and right ventricular impulse, are, the former occasionally, the latter frequently, found as coexistences, in consequence of dilated hypertrophy being gradually produced in those situations. The pathological reason of the left auricular enlargement is sufficiently clear: the right ventricle may be supposed to grow thick, through its long sustained effort both to get rid of the undue supply of blood tending to stagnate abidingly within it, and also to overcome the capillary stasis within the lungs.

The area of percussion-dulness, especially to the right side, will on similar grounds be increased. But it must be remembered that right dilated hypertrophy may possibly be deficient, where mitral stenosis is demonstrated after death, and where clinical, as well as anatomical, reasons are to be found for believing the valvular defect to be of old standing.

611. *State of the pulse.*—There is nothing distinctive in the

characters of the pulse, so long as the mitral constriction remains pure; the "smallness" which has been theoretically assigned to the arterial beat, though occasionally to be noted, is certainly not necessarily present. The pulse may, in a word, be perfectly free from significant change in frequency, in strength, or in fulness. The beat, when regular in repose, may become irregular under exertion. In cases of abiding irregularity there is either mitral reflux, dilatation, or (and this is probably the main cause), neurotic disturbance of the rhythmic motor-centres. And it must not be forgotten that mitral stenosis tends to regularize the irregular pulse of mitral insufficiency,—probably by lessening the quantity of blood, the ventricle is called on to dispose of at each systole [607]. (*Vide* APPENDIX.)

612. *Effects on the capillary systems.*—(*a*) *Systemic.*—Mitral stenosis seems even slower in producing systemic congestion and dropsy than insufficiency of the valve. In a certain proportion of cases collected by Hilton Fagge,* embolic impaction had occurred in a lower limb,—the plug, being large enough to obstruct the main artery, had, he reasonably conjectures, rather separated from a thrombus in the left auricular appendix than from valvular vegetations.

(*b*) *Cerebral.*—Accidental thrombosis, primary or embolic, occasionally occurs within the brain; very probably less often than in cases of mitral reflux.

(*c*) *Pulmonary.*—The various pulmonary conditions, anatomical and symptomatic, ascribed to the influence of reflux, may be expected to occur in stenosis of the orifice also. The relative frequency with which the two kinds of defect act gravely on the lung-circulation is undetermined: much must of course depend on the relative amount of constriction and of insufficiency. Peacock maintains that, inasmuch as the pulmony vessels undergo gradual dilatation in cases of stenosis, pulmonary apoplexy and hæmoptysis do not readily occur.† Hyde Salter believed mitral stenosis more effective than any other cardiac disease in producing hæmoptysis.

613. There seems some reason to suppose that mitral stenosis ranks among valvular læsions the least often dependent on antecedent rheumatism.

MITRAL STENOSIS AND INSUFFICIENCY COMBINED.

614. When stenosis is carried to a high point, insufficiency of

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* Guy's Hospital Reports, series iii., vol. xvi.

† Med. Chir. Rev., vol. 40.

the valve proves an occasional coexistence. As in the case just referred to [609], very great shortening of the cordæ tendineæ then acts as the common cause of insufficiency.

615. When the two anatomical conditions are associated, double murmur may be heard, systolic and præ-systolic. Or the præ-systolic portion may be inaudible, especially if the heart's action be weak. In this case audibleness in the vertebral groove will coincide with rhythm and other characters in demonstrating, it is the regurgitant division of the two murmurs that is retained. But the regurgitant may be the deficient murmur of the two; and it occasionally happens that now one, now the other, becomes inaudible. Skoda appears to hold that when the two physical imperfections exist, there are *always* two attendant murmurs,—the apparent singleness of murmur in some cases being an illusion dependent on the two murmurs following each other so closely as to simulate continuousness, while the quality and pitch of both are indistinguishable. This view may be contested on two grounds. In the first place, the quality and pitch of the two murmurs are confessedly on all hands different as a rule,—it is a pure hypothesis to maintain they are sometimes identical. In the second place, as, occasionally, no murmur has been heard during life, where mitral stenosis alone has been found in a well defined fashion after death, why should not its own murmur be sometimes deficient, where stenosis is associated with insufficiency? [118].

As a rule the two murmurs are not only distinguishable in front by their time and dividing pause, but by their quality and pitch.

615. *State of the pulse.*—The state of the pulse, as can be gathered from what has been already said [607, 611], may prove, in rhythm, dominantly that of reflux or of stenosis. Very probably, the greater the stenosis, the more regular will be the beat.

MITRAL OBSTRUCTION AND INSUFFICIENCY ALTERNATING.

616. As we have already seen [119, *b*], a form of disease may exist at the mitral valve, causing at one moment obstruction to the onward, at another reflux of a backward, current, at the orifice.

In the only case (quoted *loc. cit.*) in which I remember to have proved *post-mortem* the existence of this state of things, there was a want of perfect time-keeping of the murmur, or murmurs, audible from day to day and from hour to hour: their rhythm varied every now and then. Besides this they neither of them synchronized, as perfectly as usual, either with the systole, the diastole, or the præsystole. From such irregularity the existence of this alternating form of mitral disorder might possibly be divined.

TRICUSPID INSUFFICIENCY, REFLUX OR REGURGITATION.

617. *Physical signs proper.*—I have never known tricuspid reflux produce thrill; its production in systolic time is conceivable, if (rare occurrence) rough vegetations were formed on the auricular surface of the valve.

The characters of the attendant murmur, which, for reasons there assigned, is often absent, are set down in a previous place [110].

The only peculiarity of importance in the state of the heart's sounds is that the second proves habitually weak at the right apex, almost to extinction sometimes,—in all probability in consequence of the smallness of the current sent on through the pulmonary artery. In an instance of well marked tricuspid regurgitation, where the second sound was exceptionally strong at the right apex, the peculiarity seemed traceable to dilatation of the pulmonary artery just above the sygmoid valves.* Both sounds at the base and at the aortic cartilage may retain their natural characters; if the disease be pure, the second sound is weak at the pulmonary cartilage.

618. *Indirect Physical signs.*—Tricuspid reflux being commonly coincident with dilatation (simple or hypertrophous) of the right ventricle, the signs of that anatomical condition are discernible as follows:—Epigastric and right sternal impulse, of variable force, but out of proportion with that on the left side, unless the left ventricle be accidentally hypertrophous also, and increased area of percussion-dulness, traceable mainly to the right. Hypertrophy of the right ventricle will prevent the enfeebling of the second sound appertaining to the pure regurgitation.

619. *State of the pulse.*—Those arteries, the pulmonary, which tricuspid insufficiency may be supposed to affect directly, cannot be felt; and there is nothing peculiar in the pulse of the branches derived from the aorta. The state of the pulse will, therefore, be the exponent, not of the valvular defect, but of its coexistences or results.

620. *State of the cervical veins.*—The cervical veins distended, thickened, knotty, pulsatile, and refilling readily from below, may

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* Hishin, U. C. H., Females, vol. xi., p. 286, or Med. Times and Gaz., Feb. 14, 4857.

be the seat of thrill [228—233]. But all these various signs are only found prominently in well marked cases; if the regurgitation be slight, or if the right ventricle be very weak, venous pulsation, and thrill especially, may be altogether absent. Nay, even if the right ventricle, and especially its infundibulum, be strong, these signs may still be wanting, as was the fact in the case of Hishin, just referred to.

621. *Effects on the capillary systems.* (*a*) *Systemic.*—That in tricuspid reflux the systemic capillaries are the primary and immediate sufferers, has been very satisfactorily shown by Blakiston. The student must, however, bear in mind that the relationship between tricuspid regurgitation and grave systemic stagnation is not so intimate and essential that, given the former, he may take it for granted the latter must, *e necessitate rerum*, follow [512]. And I further believe that the concomitant dilatation of the right cavities plays a very essential part in generating stagnation and dropsy, where these actually occur. The systemic effects are the same as those occurring in dilatation, and in the Section on that disease their description will be found [523].

(*b*) *Pulmonary.*—The pulmonary capillaries not only escape primary congestion in this form of disease, but may be considered theoretically to be under-supplied with blood. Be this as it will, however, they do in the course of time undergo secondary engorgement in sequence to systemic obstruction.

(*c*) *Cerebral.*—Blakiston's results tend powerfully to show that obstruction of the cerebral capillary vessels and apoplexy are much more frequent in cases of heart-disease with, than without, stagnation of the systemic capillary circulation: hence the inference, that cerebral obstruction and apoplexy are more connected with the heart through general cardiac dilatation and tricuspid regurgitation than through hypertrophy of the left ventricle [493]. It is to be understood that no reference is intended here to the effects of accidental thrombosis or embolic obstruction within the brain (both of them rare, as attendants on tricuspid reflux), but merely to the effects of capillary mechanical stasis.

(*d*) *Hepatic.*—The conclusion has been suggested to me by a small number of facts, that tricuspid regurgitation plays a certain part in generating cirrhosis of the liver,—difficult though it may be to readily understand how the necessary influence is exercised through obstruction in the circulation of the hepatic veins. That jaundice is sometimes induced through such obstruction, is indubitable.

(*e*) *Reno-genital.*—Congestive albuminuria, as also engorgement and œdema of the female pudenda, with passive leucorrhœa, are much more frequently met in connection with the present than with any other condition of valvular disease.

622. It may on first thought appear strange that such serious consequences should be ascribed to tricuspid regurgitation,—a phenomenon which the surmise of John Hunter, the arguments of Adams, and the experiments of W. King, would lead us to suppose exists in health, as a normal provision against overloading of the pulmonary capillaries.

But does such normal regurgitation really exist? Valentin denies it.* Hope conceived that the absence of tricuspid regurgitant murmur in healthy persons sufficiently disproved the possibility of any such regurgitation: his argument was, however, valueless, for, as is well known, even morbid regurgitation, highly marked, may exist without any murmur. Still I agree with Hope in his refusal to admit the reality of regurgitation in health: my grounds are, that if regurgitation existed, it would visibly affect the venous circulation in the neck,† and that the experiments on which the doctrine is founded do not represent what common sense leads us to suppose must be the state of action at the tricuspid orifice during life. Besides, if, as is the fact, the valve, provided itself and the orifice be perfectly natural, will, after death, support water poured into the ventricle from the apex, how can we admit that during life the mechanical action shall be less complete? Far from this, there is all reason to admit that it would be more complete in virtue of the instinctive faculty of closure which, the experiments of Kürschner seem to prove, exists in the orifices [230, *note*]. Conceding, however, *argumenti gratiâ*, that slight and occasional regurgitation is a normal fact, it might nevertheless be true, as actually is the case, that highly marked and permanent regurgitation should act as the source of most serious disorder.

TRICUSPID STENOSIS, CONSTRICTION OR OBSTRUCTION.

623. *Physical signs proper.*—Constriction of the tricuspid orifice would theoretically give the diastolic, or rather præ-systolic, murmur, of maximum force at the ensiform cartilage, already dis-

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* Physiologie, Bd. i., S. 425.

† The case of Hishin above referred to, however, makes me now doubt the real justness of this argument.

cribed [114]. But this condition of the orifice is excessively rare, because atheroma and calcification are themselves extremely uncommon in this situation; while, on the other hand, an amount of coarctation reducing the orifice to the size of the middle finger may not, as a case observed by Hope proves, give rise to murmur, —probably on account of the weakness of the current.

624. The following extract from my case-books seems to furnish the characters of tricuspid obstruction and regurgitation with preciseness, but there was no *post-mortem* examination to settle the point decisively:—

> "Wm. Hodson, ætat. 48; anasarca; constant cough, with rather profuse mucopurulent expectoration; no albumen in urine. Heart's apex beats feebly in fifth space; its deep-seated dulness does not reach more than an inch to the right of the sternum. Both external jugulars considerably swollen, the right highly pulsatile from below, the left less so, though a small communicating vein, crossing the clavicle, pulsates slightly. At *mitral apex*, no murmur; first sound feeble, second sound clear, full, and accentuated. At *tricuspid apex*, that is precisely at point of left costal angle [12], there is a soft double murmur, the diastolic division much better marked about level of the fourth interspace. At the mid-sternal base, just below the level of the third cartilages, it cannot be said that there is any murmur. The second sound at the tricuspid apex is frequently reduplicate, very rarely so at the mid-sternal base: the diastolic-timed murmur is louder and more prolonged than the systolic. Pulse visible in posterior tibials and radials, not in femorals. No cervical hum. . . . . In sitting posture second sound almost constantly reduplicate at tricuspid apex, whereas in at least twenty beats only once reduplicate at base, and not once at mitral apex (U. C. H., Males, vol ix., p. 64—67)."

The heart not having been lowered in position, it would on that ground alone be unjustifiable to refer the diastolic murmur, heard at the left costal angle, to aortic regurgitation: besides the absence of murmur at the actual base settles the question against the aortic origin of either systolic or diastolic murmur.

In a case recorded by W. T. Gairdner* præ-systolic murmur "beginning immediately after the second sound contained (*diminuendo*) throughout the pause, and thus went on (*crescendo*) up to the first sound, at which it stopped abruptly," and audible "over the tricuspid orifice," appeared to that cautious observer to indicate tricuspid obstruction, "with all but mathematical certainty." I learn from himself that the patient furnishing this observation died in April, 1872; when, on examination, "not only was there obstruction of the tricuspid orifice, but obstruction (without even the slightest flaw in the valve) by a tumour attached to the auricle, and floated down upon the orifice after the manner of a ball-valve."

625. Actual experience of this form of valvular defect, espe-

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* Clinical Medicine, p. 602, 1862.

cially in an isolated state, is too limited to furnish a reliable account of its symptomatology.

It may, however, be assumed on existing evidence that the jugular veins will be more or less distended and pulsatile; that systemic mischiefs will be slower in occurring than in cases of reflux at the same orifice, and that very fair health may be maintained long after the existence of stenosis has become demonstrable.

AORTIC STENOSIS, CONSTRICTION OR OBSTRUCTION.

626. *Physical signs proper.*—In well marked cases, especially if the orifice be the least rough, systolic basic thrill may be felt.

The essential sign of aortic stenosis is systolic basic murmur possessing the attributes already set forth [111]. But, as will there be seen, this murmur is variously imitated by murmurs of other mechanisms; while weakness of ventricular action, or extreme smoothness of the constricted orifice, may prevent the development of murmur in well marked stenosis. Excitement of the heart by quick movement may remove the latter difficulty; the former dilemma is to be escaped from in some cases by carefully considering the diagnostic claims in each individual instance of all possible sources of such murmur and eliminating them one by one.

The natural first sound of the heart remains audible at the left apex, though more or less covered by the basic murmur: the second sound, weak at the apex, at the base, and at the aortic cartilage, may be slightly murmurish at the two latter points, in consequence of some regurgitant tendency. The second sound is occasionally reduplicate at the base. The systolic murmur may be prolonged enough to cover the second sound more or less completely at the base; the latter may then, as a rule, be caught at the apex.

627. *Indirect Physical signs.*—The impulse and altered percussion-sound characteristic of hypertrophy, with or without dilatation, of the left ventricle, are the commonly coincident signs. A rare instance every now and then occurs, wherein the murmur of mitral insufficiency is generated through dilatation of that orifice following on dilatation of the ventricle, which, in turn, has been entailed by the enduring obstruction at the mouth of the aorta; besides this, insufficiency of the aortic valves themselves is often present as an independent effect of a common cause.

627. *State of the pulse.*—The pulse, in cases of moderate coarctation, is not materially affected. If the constriction be great, the pulse, though regular in force and rhythm, is small, hard, rigid, concentrated, and prolonged; hardness and force signify hypertrophy behind the narrowed orifice. If there be no such hypertrophy, the pulse may be soft, quasi-undulatory, and make but little impression on the finger.

Sphygmograms of the pulse in pure aortic constriction have not appeared in great number: the pure affection is in truth sufficiently rare. Marey, inferring that the upstroke must be longer than natural from the prolonged effort of the blood to pass through the narrowed orifice, attempted to show that a curve would replace the usual vertical line at the commencement of the pulsation. His tracings appear to lend but faint support to his theory. Lorain, however, gives some drawings which exhibit a slow and oblique line of ascent, with secondary curve and rounded apex. (*Op. cit.* p. 272.) If marked hypertrophy of the left ventricle coexist, the line of ascent may be vertical, despite the obstruction. (*Vide* APPENDIX.)

628. *Effects on the capillary systems.*—(*a*) *Systemic.*—It is truly remarkable to what an extent this coarctation may be carried, without producing systemic stagnation: the opening may be no larger than a pea, without leading to the very slightest œdema, even of the ankles. This immunity, however, only holds, as long as the capacity of the ventricles and the width of the tricuspid orifice remain unaffected; if the blood becomes spanæmic, too, anasarca occurs independently of these latter changes. It has been conjectured that the peculiarity depends on the slowness of the circulation in old people; but the circulation is not always slow in old people, and young adults, with constriction of the aortic orifice, also remain free from systemic dropsy.

(*b*) *Pulmonary.*—Aortic constriction exercises no direct effect on the pulmonary capillaries; some indirect tendency to stagnation arises from the difficulty experienced by the blood, flowing from the pulmonary veins, in its entrance into a ventricle, which, again, has itself great difficulty in discharging its contents. Oppression of chest, dyspnœa and congestive symptoms, to a slight extent, may thus be produced.

(*c*) *Cerebral.*—It seems theoretically probable that a constricted aortic orifice will weaken the impulsive effect of a hypertrophous left ventricle on the brain. Embolism, though less frequent than in mitral reflux, is to be looked for as an occasional accident.

AORTIC INSUFFICIENCY, REFLUX OR REGURGITATION.

629. *Physical signs proper.*—Diastolic basic thrill is occasionally to be felt, independently of spanæmia [63, *a*]. The murmur of aortic insufficiency has already been sufficiently described [115]. Some few observations will there be found on the different morbid states capable of engendering the murmur. I have scarcely met with any condition simulative of true reflux murmur, except that caused by roughness with dilatation of the ascending portion of the arch; the condition of the superficial pulses will distinguish the cases. Besides this, it is conceivable that pressure on the outer surface of the aorta just above the valves, might interfere with the closure of these, and so produce regurgitant murmur: thus in a case of displacement of the heart by pleuritic effusion, diastolic murmur existed under circumstances tending very positively to connect the murmur with the displacement.* I have never encountered a positive example of aortic *blood-murmur* diastolic in time, but such a murmur may be simulated in the following way:—spanæmia exists, with a strong systolic basic murmur; at the same time, deep-seated hum is present in the pulmonary veins; this hum is covered during the systole by the strong, blowing aortic murmur, but becomes audible during the diastole, when there is no aortic murmur to interfere with it.

Aortic regurgitant murmur is, as far as I know, constant, where its physical cause exists; weakness of ventricular action obviously cannot have the same effect in rendering a regurgitant, as a direct, murmur obscure. If a double aortic murmur exist, the systolic portion is best transmitted upwards in the course of the vessel, the diastolic generally downwards in the line of the sternum,—the latter peculiarity probably owing to the direction of the current. The infrequency of direct tricuspid murmur, and the coexistence of other signs of aortic regurgitation, will commonly prevent any error arising from the loudness of the aortic murmur at the ensiform cartilage. I can, as a bare possibility, conceive complete destruction of the aortic valves, with smooth surface, to exist without murmur; but have never observed the fact [633].

State of the heart's sounds.—The state of the heart's sounds is as follows:—The first, at the base, may be natural or very nearly so, dull, obscured by a soft murmur, or masked totally by a harsh one: or it may be inaudible at the base, and well defined at the mitral apex; while in some rare instances it cannot be heard at all.[*] The second sound, at the left apex, may be null, faint, or distinct and sharp; in the latter case, the sound heard is probably that of the pulmonary valves transmitted,—scarcely that of fall of the aortic blood, during regurgitation, into the ventricle below [98†]. At the aortic cartilage the first sound is of variable character; the second replaced by a murmur, more or less marked. Both sounds may be natural at the pulmonary cartilage, and occasionally the second be accentuated, though there be no mitral regurgitation: in some instances, however, the basic diastolic murmur is very loudly conducted to this cartilage, covers the second sound there, and *pro tanto* simulates the essential sign of insufficiency of the pulmonary valves.

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* H. Morris, U. C. H., Males, vol. vi. p. 35.

630. *Indirect Physical signs.*—The concomitant signs are those of hypertrophy, eccentric or simple, of the left ventricle; frequently of aortic constriction, and occasionally of mitral regurgitation. When hypertrophy is highly marked, the heart's shock is singularly abrupt, and followed sometimes by a fall back so sudden, as to simulate by its jog a diastolic impulse.

631. *State of the pulse and arterial system.*—The pulse is sudden, abrupt, short, jerking—a distinct fall back instantly following the rise of the vessel. Commonly perfectly regular in rhythm, more or less notably lagging behind the systolic sound of the heart [103, *i*], occasionally bisferiens, each pulsation is, in rare instances, attended with thrill. Thrill may be powerful in the subclavian artery, while very feeble in the radial, indeed only producible by artifice [196]. The radial pulse is further characterised by various of these qualities being intensified through raising the arm; and the beat of the superficial arteries (as well as some of the deep-seated class) is visible and locomotive [141, *b*].

To what extent may the visible and locomotive pulse be trusted to, as evidence of aortic regurgitation? In the first place, no case of that disease, well defined in *all* its other clincal characters, has ever fallen under my notice in which visibleness of the superficial pulses was not more or less obviously present. But I have known murmur, basic and diastolic (and obviously from its concomitants not seated in the pulmonary artery), where the radials were not visibly pulsatile, though the patient was thin.[*] Such cases are very rare; but they certainly do constitute exceptions to the rule. In the second place I have never observed *highly marked and extensive* visible pulsation without aortic regurgitant disease. Hope thought that, where there was mitral regurgitation or contraction to any amount, aortic regurgitation failed to render the pulse visible. This seems somewhat theoretical, and is positively opposed to what I have seen.[†] But in the third place, in aged persons of thin frame, whose vessels are calcified and tortuous, a slight degree of the phenomenon may certainly be noticed in the smaller arteries, independently of any affection of the aortic valves; and if the left ventricle be hypertrophous in such persons, the amount of visibleness will be materially greater; but still, as far as I have seen, will fail to affect the larger trunks. I have very rarely[‡] known simple or dilated hypertrophy alone produce this sign in persons under the age of forty, whose limbs were even moderately well covered with flesh. If this qualification regarding age be borne in mind, exceptional cases will, I think, be found to be singularly few. In the fourth place I have occasionally seen cases of marked emphysema, unattended with the least clinical evidence of aortic disease, where the majority of the superficial arteries, even the femorals, pulsated visibly.[§] In the fifth place, in a case which presented all the clinical requirements for the diagnosis of coarctation of the arch of the aorta, the diastolic heart-sound being healthy, several of the superficial arteries were visibly locomotive.[‖] Sixthly, it may be added that aneurismal varix of the aorta and pulmonary artery will likewise produce such visibleness [815, 818].

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[*] In some of these rare cases of absent first sound the mitral valve has proved healthy on *post-mortem* examination: how, on the valvular theory of that sound, is its deficiency to be explained? Cockle (Insufficiency of Aortic Valves, &c., p. 25) adopts a hypothesis of Traube, infinitely fine-drawn, the basis of which, "an elongated flattened, atrophous, cirrhotic state of the papillary muscles," seems of doubtful reality, seeing that Gendrin teaches the papillary muscles and mitral valve undergo hypertrophy in aortic reflux. The absence of first sound under the circumstances would appear to me to afford an accessory proof that the mitral valve has nothing to do with its generation [95]; and that the sound is wanting because the impulse of the ventricular blood against the base of the aortic column cannot take place in the ordinary way, in consequence of the perturbation in the normal manner of blood-motion caused by the reflux.

[†] In addition to what is said, in the place referred to, on the possible generation of an imitation of natural second sound by fall of blood from the aorta against the inner surface of the apex of the ventricle, it occurs to me to suggest whether, if the phenomenon be ever really sonorous, the noise is not more likely to possess the characters of a murmur than of the diastolic sound of the heart.

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[*] S. Dunn, U.C. H.. Females, vol. xvi., p. 296.

[†] Denham (U. C. H., Males, vol. vi., pp. 69—78) had marked mitral regurgitation and constriction associated with aortic regurgitation, yet his radial, brachial, posterior tibial and femoral pulsations were well visible.

[‡] Beere, U. C. H., Females, vol. x., p. 98. Ætatis 40,—visible pulses, hypertrophy of left heart, without diastolic basic murmur.

[§] Slater, U. C. H., Males, vol. xii., p. 79; neither aortic regurgitation nor hypertrophous heart, ætatis 44.

[‖] Gunnee, U. C. H., Males, vol. xiv., pp. 38. This will be fully spoken of in a future place [837].

In all these exceptional cases the symmetry of the phenomenon has appeared to me to be less perfect, than when dependent on aortic regurgitant disease.

Another character of the blood-course in the arteries is that its impulse is retarded,—the pulse in the radial lagging behind time more or less notably [103, *i*].

Some years ago Duroriez* described double intermittent blowing murmur in the femoral artery, as a very characteristic sign of aortic reflux; pressure being applied to the vessel, a systolic murmur becomes audible on the cardiac side, a diastolic on the systemic side of the point compressed. The French observer maintains this femoral murmur proves, *quoad* diagnosis, all the more valuable, as it may sometimes be detected, when, "owing to disturbance of the proper relation between the contractile force of the artery and the aortic orifice," the murmur of reflux is deficient at the base of the heart. As to the occasional existence of this form of double artificial arterial murmur (in various other arteries, too, as well as the femoral) no doubt can be raised; but it is not pathognomonic of aortic reflux. As stated in former editions of this work, even hæmic murmur of double time may occasionally be heard in the femoral and other vessels under the influence of pressure either artificial [224], or produced by disease [225].

The tactile characters of the pulse are so peculiar and well defined that it would be strange, if its graphic representation failed to display something characteristic. The typical characters appear in a line of ascent vertical and high; pointed and notched apex; oblique line of descent. But it must not, on the other hand, be assumed that these sphygmographic characters are pathognomonic of aortic reflux; the pulse in old age, and in people with atheromatous arteries, as fairly admitted by Lorain, exhibit almost identical peculiarities; and Saunderson gives tracings appertaining to mitral reflux and to aortic reflux (*loc. cit.*) of accurately similar types. And on the other hand, palpitation and certain other circumstances may seriously modify all special characters of the aortic sphygmogram.

632. *State of the capillary systems.* (*a*) *Systemic.*—Aortic reflux may be carried to a high degree, as shown by the well defined characters of its murmur, its peculiar tactile pulse and its visible and locomotive arterial beat, and exist in this state for years, without giving rise to obvious systemic disturbances of any

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* Brit. and For. Med. Chir. Rev., July, 1861, p. 259.

kind. Especially does this statement hold true of œdema of the extremities.*

(*b*) *Pulmonary.*—Aortic regurgitation affects the pulmonary circulation, either indirectly through mitral regurgitation (of which it is itself the original cause), or more directly through the embarrassment produced by the collision of blood, falling backward from the aorta, with blood coming forward from the auricle. The tendency to sequential hypertrophy of the left ventricle, which sometimes arises mainly about the apex in these cases, does not afford as great help to the circulation as in aortic stenosis; on the contrary by increasing the amount of distension of the aorta at each ventricular systole, it intensifies the force of the succeeding recoil, though, probably, by tending to prevent systemic stasis it may, to a certain extent, prove serviceable [662, *g*].

(*c*) *Cerebral.*—The cerebral capillaries can only be affected secondarily through the pulmonary class; and the systemic capillaries are much in the same position.

All three classes of capillaries, systemic, pulmonary, and cerebral, may escape for years after the establishment of reflux.

Aortic Stenosis and Insufficiency Combined.

633. *Direct physical signs.*—Double basic thrill, systolic and diastolic, may be looked for in some cases; I do not chance to have met with it.

634. The essential sign is double basic murmur; the systolic portion sometimes higher-pitched and shorter than the diastolic; while in other instances a long, loud, and rough systolic is followed by a feeble soft diastolic murmur; or, again, the systolic portion may be much the longer at the base, the diastolic at the ensiform cartilage. The two portions, commonly separated by a distinct pause, may, as it were, run into each other.

But the systolic portion may be wanting, though stenosis is distinctly visible after death; while, on the other hand, if the aorta, close to the valves, be at all roughened by disease, and especially if it be bulged in that spot, a systolic murmur, indistinguishable from that of stenosis at the orifice, may be heard.

And, again, the diastolic portion of the double murmur may be masked. Thus, in a case of aortic constriction, with systolic basic murmur, and with visibleness of the superficial pulses carried to the highest degree, the systolic murmur was so prolonged as to cover completely any diastolic murmur that may have really existed: but the diagnosis of aortic insufficiency, made on the ground of the extreme visibleness of the pulses, was proved to be perfectly correct at the *post-mortem* examination.

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* *E. g.*, Barnes, U. C. H. Males, vol. v., p. 216; but the fact is a common one: I have known this absence of dropsy, where the signs of regurgitation had existed for at least three years.

Further, the systolic division may increase in intensity, as the stethoscope is carried up the arch, while the diastolic dwindles so as to become well-nigh inaudible,—the exact reverse arrangement of changing intensities may be traced down the sternum.

634. *Indirect Physical signs.*—They are those of dilated left hypertrophy.

635. *State of the pulse.*—It might theoretically be supposed that the special pulse of aortic regurgitation [631] would be deficient, if stenosis of the same orifice were carried to any amount. Clinically I have found the fact otherwise: even with extreme stenosis the reflux characters have been retained.

AORTIC INSUFFICIENCY REPLACED BY STENOSIS.

636. Of this rare event, the possibility of which was, I believe, first demonstrated in the edition of 1863, the following solitary example has fallen under my notice:—

"December 7, 1860. Mr. . . . , ætatis 57; in good condition; sleeps badly, but with equal comfort on both sides; no dreams; can walk *five* miles an hour with ease; walked forty-two miles in nine hours and forty minutes a year ago; can walk up hill; complains specially of a disagreeable sensation at and below the left nipple, not amounting to actual pain, nor attended with faint feeling, and relieved by pressure; once felt a sensation, after a violent pedestrian effort, as if something had given way within the chest, and exclaimed, "Good God, I wish I had not done that." Never had rheumatic fever nor gout; pulse said to have been irregular from birth; certain that it was ascertained to be so at ætatis three; pulse now irregular, of medium frequency, not visible at radials, at one ulnar only, other arteries not looked at.

"Heart examined in standing and sitting postures: action somewhat too diffused, and impulse too forcible; deep-seated percussion-dulness slightly in excess; medium-pitched, prolonged, blowing, diastolic, basic murmur, rather louder at the top of the sternum than at the precise base; slightly less resonant under percussion at the right than the left sterno-clavicular angle, also at the left than the right interscapular region,—no pain in either of these situations. From these facts the diagnosis followed: moderate dilated hypertrophy of the heart: aortic regurgitation; calcification of the arch of the aorta [223]; and possibly some slight dilatation of the arch. I never saw the case, in life, again.

"This patient died at the beginning of April, 1861. I then learned that he had, from time to time been seen by Sir T. Watson, in whose notes the existence of diastolic murmur at the base had been recorded as early as June, 1860. Sir T. Watson saw him for the last time in February, 1861, and then to his surprise ascertained that of the diastolic murmur no trace impressed the ear, while a basic systolic murmur had now become loudly audible. The mystery, we found, received an easy solution in the state of the aortic valves. The substance of these valves had been considerably destroyed,—to such extent as to admit of free reflux. But to the edges of the injured valves were attached masses of very soft fibrine, of such bulk as to block up the orifice of the vessel completely, when looked at from above. Obviously this thrombal plug, of relatively recent formation, must at once have prevented regurgitation and opposed a formidable barrier to the direct current from the ventricle. There was besides a recent perforation of the long tongue of the mitral valve, which may possibly, though to a considerable extent plugged with fibrine, have intensified systolic murmur. The arch of the aorta was slightly dilated, atheromatous but not calcified; the left heart slightly enlarged and hypertrophous." *

PULMONARY STENOSIS, CONSTRICTION OR OBSTRUCTION.

637. *Physical signs proper.*—Systolic basic thrill reaching to the second cartilage, in association with the systolic basic murmur, already described [112], are the essential physical signs. Except in cases of cyanosis this murmur is of excessively rare occurrence.

Its mode of propagation ought to distinguish a pulmonary from an aortic systolic murmur, and so it commonly will. But in some cyanotic children the loudness of the murmur makes this character difficult of substantiation. Hope taught that a pulmonary constrictive murmur seems closer to the ear than an aortic, and on a "higher key, ranging from the sound of a whispered *r* towards that of an *s*:" the reason being that the pulmonary artery is nearer to the surface of the chest than the aorta. As regards closeness to the ear, his statement is, doubtless, correct; as regards higher pitch, the reason assigned for this is obviously erroneous; and, indeed, Hope himself unconsciously admits this, by adding that he has known the murmur "fall below *r*, when the circulation was feeble and slow and the obstruction slight."

638. *Indirect Physical signs.*—Supplemental hypertrophy with dilatation of the right ventricle ensues in course of time, and is well seen in some cases of cyanosis.

639. *State of the pulse.*—The absence of special character gives distinctiveness to the radial beat.

640. *State of the capillary systems.* (*a*) *Systemic.*—Venous stasis does not occur in childhood with the grave results that might be anticipated: true, venous discoloration of skin and bulbous malformation of the finger-ends are carried to an extreme amount, but anasarca is slow in occurring.

(*b*) *Pulmonary.*—The pulmonary system must be ill supplied with blood: hence the subjective sense of want of breath and easily evoked fits of dyspnœa appertaining to the disease.

PULMONARY REGURGITATION, REFLUX, OR INSUFFICIENCY.

641. *Direct Physical signs.*—The theoretical characters of the

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* Another case, illustrating this interchange of murmurs at the aortic orifice, has recently been published by T. W. Gairdner, Brit. Med. Journ., May, 1872.

murmur significant of this state will be found elsewhere [116]. Basic diastolic thrill may occur.

642. *Indirect physical signs.*—The absence of jugular distension and pulsation, as well as of the peculiar characters of aortic regurgitant pulse [631], will give important negative evidence as to the origin of the murmur.

643. *State of the capillary systems.*—Theoretically, the effects on the systemic and cerebral capillary circulation must be most serious; and a sensation of dyspnœa, arising from the smallness of the quantity of blood actually reaching the lungs by each systole, might, unless the force of habit would counteract this influence, be expected.

644. Very few examples of pulmonary insufficiency, observed during life and after death, have been recorded in this country. In truth, I only know of one,—that described by Gordon.* A boy, aged twelve, admitted into hospital with cold surface, feeble palpitation, very weak pulse, cough, copious expectoration and moist rhonchus, had thrill over the entire cardiac region, and a double murmur, loudest at the base, and inaudible at the apex, without murmur, fremitus, or visible pulsation of the superficial arteries. The thrill and "anomalous circumstances of the case" led Gordon to diagnose open foramen ovale: he was right,—a large opening existed between the auricles. But the pulmonary valves, thickened and shortened, allowed a column of water to pass through; the other valves were generally healthy.

Unfortunately the rhythm of the thrill is not mentioned. It would be opposed to our experience of the parallel aortic affection, to suppose thrill easily produced by pulmonary regurgitation; the sign probably depended on the obstructive influence of the diseased pulmonary valves, and would have been systolic in time.

A loud musical diastolic murmur (where most distinctly audible and how transmitted, we are not informed) was noted by Stokes, in a case of incompetence of the pulmonary valves, caused by widening of the orifice, the valves being sound: there was no aortic insufficiency.

In a case (seen, however, only in life) by Eddison and Fothergill, the symptoms and signs noted were as follows:—

"Subjective: dyspnœa on any exertion, general debility, and incapacity for bodily labour. Objective: considerable increased dulness over the right ventricle and auricle, extending from the second right costal cartilage over the third and fourth, and from the base of the ensiform cartilage to the right apex; also a soft, blowing diastolic murmur heard from the third left costal cartilage to the base and towards the xiphoid cartilage." *

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* Vid. Stokes on Dis. of the Heart, p. 166, 1854.

PULMONARY STENOSIS AND INSUFFICIENCY COMBINED.

645. *Direct Physical signs.*—Thrill may be felt of systolic rhythm at the third left cartilage; conceivably the thrill might be double. In a well defined case, recorded by Begbie, double murmur was perceived at the same cartilage: the systolic portion, blowing and unusually loud, extended over the whole upper chest, but scarcely to the carotids; the diastolic, much less intense, scarcely travelled beyond the cartilage in question.†

646. *Indirect Physical signs.*—The heart may continue of normal size for years, as was the fact in the case just referred to.

647. *State of the pulse.*—No special character of any kind.

648. *State of the capillary systems.*—(*a*) *Systemic.*—There need be neither lividity of face nor œdema of the feet.

649. (*b*) *Pulmonary.*—Breathlessness, more or less marked, may prove the sole symptom, unattended with cough, hæmoptysis, or expectoration.

PHYSICAL DIAGNOSIS OF VALVULAR DISEASES IN GENERAL.

650. In fixing on the seat of production of any given murmur, the first point for the observer to establish is the position of the heart itself: if this be abnormal, as it very often is in consequence of changes in the ventricles, preceding, coadvancing with, or sequential to the existing valvular disease, allowance must be made for the influence of such displacement in altering the maximum position of murmurs. Now, the general tendency is to some form of enlargement of the organ; and, as enlargements lower it, the maxima points of murmurs are very commonly slightly lower than the maxima points of the corresponding sounds in health. Certain conditions of the aorta or pulmonary artery, of the lungs, pleura, or mediastinum, also throw murmurs into unnatural sites, by changing the position of the heart. Pleural fluid accumulations have more influence in transposing the audible point of apex, than of basic, murmurs; because, while detruding the heart as a whole sidewards, these accumulations push the apex further away than the relatively fixed base.

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* Brit. Med. Journal, Dec. 17, 1870.

† Beale's Archives, vol. ii., p. 12, 1860. Patient died, ætatis 20, from fracture of the skull through a fall: there were four pulmonary valves, one of them congenitally imperfect.

651. The state of the heart's substance also may exercise a direct influence on the intensity of murmurs. Thus hypertrophous texture, lying, in the course of the circulation, behind a murmur, will intensify this, if it be direct; exercise little effect on it, if it be regurgitant: take the instance of an hypertrophous left ventricle with severally a constrictive and a regurgitant aortic murmur. Hypertrophous texture, lying in front of a murmur, is without effect upon this, if it be direct; intensifies it, if it be regurgitant: take the instance of an hypertrophous left ventricle with severally a constrictive and a regurgitant mitral murmur. Dilatation and enfeebling affections of the heart's substance weaken any direct murmurs through orifices on which such substance may play. Dilatation lessens the force of a regurgitant murmur, in so far as power of backward propulsion is concerned; but, inasmuch as it allows of considerable accumulation of blood for reflux, the weakening influence is somewhat counterbalanced.

652. The physical conditions of a given murmur positively existing at a given orifice, the character of that murmur may be seriously affected by the anatomical state of other orifices. Thus, aortic constriction may be almost murmurless, if there be any great coexistent mitral regurgitation, the aortic current is so materially weakened by such regurgitation.

653. Of two murmurs, produced synchronously at different orifices, one may mask or cover the other. An aortic constrictive and a mitral regurgitant murmur may exercise this reciprocal influence; and a mitral regurgitant may completely drown a tricuspid regurgitant murmur [633]. In the interspace between the maxima points of two synchronous murmurs, however, a spot may generally be detected, where the intensity of murmur is less than at either of the pair, and the quality, as well as the pitch, different.

654. An hypertrophous and widened state of the aorta will intensify a reflux murmur at its own orifice; and if pouching exist at the sinuses of Valsalva, the rippling direction thereby given to the current will intensify both a direct and a reflux murmur.

654*. The composition of the blood, too, is all-important: spanæmia greatly strengthens the force of murmurs of all kinds. The pulmonary veins and superior cava may be the seat of spanæmic murmur, simulating cardiac murmurs, — especially systolic at the apex and diastolic at the base.

655. Again, modified conducting power of adjoining textures may lead to error: thus, an aortic constrictive murmur may be

heard better to the left than the right of the sternum, if the left lung be solid and the right emphysematous. Lastly, excessive irregularity and frequency of the heart's contractions, may make it impossible to determine the synchronism of a murmur (p. 78).

656. The physical conditions of any orifice, or of its valve, giving rise to murmur, are liable to change,—now alteration in existing murmur will or will not ensue, according to the nature of that change. Four acoustic varieties of event may be noted under these circumstances.

(*a*) The most common occurrence of the kind is aggravation of any given anatomical state, constrictive or regurgitant; such aggravation will, as a rule, exercise but little appreciable influence on the murmur, and certainly no influence which can during life be assigned with surety to its cause.

(*b*) Cases are pretty frequent in which, disease of constrictive effect existing at an orifice, the valves, becoming more gravely diseased by-and-by, permit reflux; or what is more rare, the conditions of regurgitation having taken the lead, those of obstruction follow. In either case a single gives place to a double murmur.

(*c*) The conditions of murmur both of direct and of reflux mechanism, may be present at the same time in the same valve, but so related that now one, now the other, form of murmur is alone audible. Of this peculiar condition I have already spoken [119, *b*; 601].

(*d*) The fourth occurrence, of great rarity, is that in which a regurgitant murmur wholly disappears at an orifice, giving place to an obstructive one. I believe very confidently I have observed this at the mitral orifice; but I am without the evidence of *post-mortem* examination. Of its reality at the aortic orifice the proof is recorded a few pages back [636].

657. It follows from this view that the diagnosis of valvular disease, though in the majority of cases sufficiently easy, must prove occasionally beset with various and serious difficulties,—difficulties in certain instances so numerous and so singularly combined, that it would savour more of temerity, than of experienced judgment, to venture on a positive opinion. But such cases are of rare occurrence.

SYMPTOMS OF VALVULAR DISEASE IN GENERAL.

658. The really distinctive symptoms of each form of valvular disease are to be gathered, as matters of necessary inference, from

the statements already made concerning the influence of each on the different capillary systems. There are a few functional disturbances which may be considered common, though by no means to precisely the same amounts, to all of the class.

(*a*) Thus, loss of strength and loss of ability for exertion, while, relatively speaking, the flesh is well maintained, prove very usual effects, no matter which is the valvular apparatus concerned.

(*b*) Imperfect rest, startings from sleep with affright, and dreams of more or less distressing character, indicate the disturbed circulation through the nervous centres, entailed by the mechanical difficulties in the heart. As a rule, rarely broken, patients with valvular disease lie with the head high,—the position in bed is, sometimes, rather that of sitting than of lying. Still the rule is sometimes infringed: I knew a case of enormous dilated hypertrophy with aortic constriction and regurgitation and mitral regurgitation, in which night-dyspnœa was greatly easiest, when the head was kept on a slightly lower level than the shoulders.

(*c*) Various paræsthesiæ may be felt in the cardiac region,—or actual pain varying in degree between a slight aching sensation and the intensity of genuine angina. It has been plausibly enough suggested that atheromatous, and more particularly calcified disease of the valves and orifices, by interfering with the easy stretching and retraction of those parts, may explain the different varieties of painful sensation. I feel persuaded from what has fallen under my notice, that pain is more commonly connected with disease of the aortic than of any other valves.

(*d*) Palpitation, a frequent attendant on valvular disease, is in the main rather the offspring of the coexistent states of nutrition, capacity, and innervation of the heart, than of the mischief in the valves *per se*. At least, it is certain, any given form of valvular disease may reach any given degree of development without habitual palpitation proving a necessary effect.

(*e*) Dyspnœa again may occur in any of the series, or be absent in every one of them.

(*f*) Headache, vertigo, and intra-cranial paræsthesiæ may occur in connection with any one of these valvular defects.

The student must remember that no direct relationship holds between the amount and severity of general objective and subjective symptoms and the imminence of danger through valvular mischief. He, who has none of those symptoms, may die in an instant; he whose life has become, through their variety and intensity, one abiding and miserable struggle against death, may linger on for months,—nay, by possibility for years. All will depend upon the valve affected and the manner of its disease [662, *b*].

CONDITIONS OF ORIGIN.

659. We have shown in what various forms anatomical change occurs in the structure of the valvular apparatus, or of the orifices, giving rise to impediment in the circulation, obstructive or regurgitant. What are the conditions, constitutional or otherwise, under which these various forms of anatomical change occur? These conditions may be tabulated as follows:—

I. CONGENITAL.		
II. ACQUIRED.	*a.* Intra-cardiac thrombosis.	1. Spontaneous. 2. Mechanical.
	b. Endocarditis.	
	c. Diathetic and degenerative vices.	1. Rheumatic. 2. Gouty. 3. Syphilitic. 4. Cirrhotic. 5. Oily and saline.
	d. Mechanical injury.	1. Efforts. 2. Concussions. 3. Blows.

I. CONGENITAL ORIGIN.

The history of the numerous varieties of malformation of the valves belongs rather to the provinces of Teratology and pure Morbid Anatomy, than to that of Practical Medicine. But there are two aspects under which these congenital defects become of real importance to the physician. They may give rise to physical signs in no wise distinguishable from those of extra-uterine valvular changes; and, as ably shown by T. B. Peacock,* they may (whether capable or not *per se* of producing those signs) promote, nay excite, such morbid changes as shall prove in all respects, anatomical and clinical, identical with those developed after birth.

The clinical question, one which by no means unfrequently presents itself in actual practice, is this: can we, during life, distinguish from each other,—pure congenital malformation of valves; malformation *plus* extra-uterine change; and extra-uterine disease alone? To this query the response must unfortunately be in the negative. Age, sex, occupation, duration of symptoms—taken singly or in combination—give no trustworthy assurance on the matter. But we are every now and then entitled to surmise congenital origin, where the patient is enabled to affirm positively that he has never had acute injury to the heart, that his life has passed without indications of acute thoracic inflammation, and that he is, and has been, free from the diathetic vices, that are known to impress their mark upon the valves.

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* Valvular Disease of the Heart, 1865.

II. Acquired Origin.

(*a*) Intra-cardiac thrombosis, whether occurring spontaneously, or as the mechanical result of fixture of fibrine on fissures or unevennesses of the valves, may give rise to stenotic or reflux defect.

(*b*) Endocarditis, by variously altering the smoothness, the shape, the freedom of play, the manner of relationship of the different cusps, of a valve,—by destroying its substance, and by puckering its connected cords, proves the source of impediment at the various orifices.

(*c*) 1. Among diathetic causes of valve-disease acute articular (as distinguished from chronic general) rheumatism certainly holds the first place. The intervening endocarditis is not necessarily (though probably oftener than pericarditis) an attendant on serious, rather than slight, seizures of rheumatic fever.

2. The endocardium habitually escapes mischief in gouty people. Still, saline precipitation is found, occasionally, within the valves of persons known to have been long the subjects of gout. The saline matter would probably prove, in part at least, of uric acid composition, as was the case in a gouty arterial concretion analysed by Landerer.*

3. We are sometimes led to surmise, in examining cases of valvular disease, where every ordinary cause of such affection can be excluded, where the defect is known from past examinations not to be congenital, and where the patient is tertially syphilitic, that the structural changes in the valvular apparatus may be the result of syphilitic action. But proof is yet deficient.

4. Cirrhotic thickening of the cusps of a valve, or of the tendinous cords connected therewith, is not an unfrequent degenerative change.

5. Atheroma and calcification are essentially the local expressions of a diathesis,—inflammation having no necessary connec-

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* Cyc. of Anat. and Physiol. art. Products, Adventitious, p. 89.

tion with either;[*] though, of course, local inflammation may independently precede the active development of the diathesis.

(*d*) Mechanical influences are a very rife cause of valvular disease.

1. Effort: whether excessive and instantaneous, leading to rupture or fissure of the cusp of a valve (chiefly the aortic), or tearing asunder of a mitral cord,—or considerable and long sustained, leading to nutrition-changes in the valvular texture: plays an important part in the etiology under consideration.

2, 3. Concussions and violent blows very probably prove occasionally effective in the same way. A case of railway concussion once fell under my notice, in which the sufferer, a young thin girl, had been thrown forward, the chest coming in violent contact, at the union of the upper and middle bones of the sternum, with the arm-rest on the opposite side of the carriage. Here as the demonstrated result, dissecting aneurism of the arch, with injury to the aortic valves, followed on the instant: the girl had been known to have been free from cardiac affection of any kind prior to the accident.

MORTALITY, DURATION, AND PROGNOSIS.

660. *Mortality.*—That the mortality due to valvular diseases in this country steadily maintains a high rate year after year, is as certain, as it is impossible, even approximatively, to estimate the precise amount to which they, unassisted, destroy life. The nomenclature adopted by the Registrar-General would, in itself, prove fatal to any attempt at even roughly accumulating evidence concerning the mortality from these affections; but the statistics of the subject will, under any circumstances, always remain beset with inherent difficulties.

661. *Duration.*—Nor, viewing all forms of valvular impediment as a single group, is it possible to make any statement of the least utility on the question of duration. Nay, even if any individual disease of the whole number be selected, the extremes of brief and of protracted course are found to lie so widely apart, that it becomes practically useless to strike a mean. Thus mitral regurgitation may destroy life—or at least act as the chief agent in destroying life—within a year from the first appearance of its physical signs; and yet signs as indubitable of mitral regurgitation have been known to exist in other cases for a lengthened term of

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[*] Cyc. Anat. and Physiol. *loc. cit.*, p. 88.

years without the least, or with but the slightest, attendant subjective disturbance. Thus, (as a single illustration) I have watched the way of life of a gentleman, who *twenty years ago*, under my own observation, had rheumatic endo-pericarditis, followed by a *systolic* left-apex murmur, possessing in perfection all the attributes of the essential sign of mitral reflux: he is to this hour in the enjoyment of excellent health; and, were it not for slight dyspnœa under exertion, might boast of a perfect subjective state of cardio-pulmonary function, though the mitral murmur holds on unchanged.

The amount of the valvular impediment to some extent, the diathetic state of the patient to a greater extent, but yet more the *condition of the heart's substance*, sometimes afford a clue to this difference: but often the mystery proves inexplicable. Meanwhile we must be content to register the unexplained truth, that all forms of valvular disease may, on the one hand, prove the main instruments in rapidly killing, or, on the other, remain for a well-nigh indefinite period subjectively latent.

662. *Relative gravity* (*a*).—As matter of clinical experience, the chief valvular derangements may be placed in the following descending series on the basis of their relative gravity,—that is, estimating this gravity not only by their ultimate lethal tendency, but by the amount of complicated miseries they inflict:—

Tricuspid regurgitation;
Mitral regurgitation;
Mitral constriction;
Aortic regurgitation;
Pulmonary constriction;
Aortic constriction.

Of tricuspid constriction and pulmonary regurgitation too little is known practically to warrant us in assigning them their position in the series; but the two cases referred to previously [624, 644] show positively, that one and the other defect may long exist without giving rise to grave inconvenience.

The positions severally occupied by mitral constriction and regurgitation would be reversed by some observers; as far as my own experience may be taken as a guide, I have no hesitation in adopting the arrangement given above. It must be remembered, too, that tricuspid reflux is very rare as a primary affection,—and one to be looked on practically, as the final term of evolution of various cardiac affections.

(*b*) But there is a very important aspect in which the relative danger of these affections would place them in a widely different order,—I mean that of their relationship to sudden, really sudden or instantaneous, death. Taken as a group, valvular impediments cannot fairly be cited as frequent causes of sudden death; but there is one among the number of which the tendency to kill instantaneously is so strong, that the fact must always be borne in mind in estimating its prognosis,—and that one is aortic regurgitation. The first example of the kind, which drew my attention to the subject, occurred in 1852; since that time I have observed certainly eight, and probably eleven, similar cases. The manner of death is clearly syncopal; but the immediate mechanism, whether mechanical or dynamic, is difficult enough of comprehension. I have known death take place during the acts of walking, of eating, of speaking,—while the patient was emotionally excited, and, *per contra*, at a moment when he was perfectly calm. And a very singular proposition, which flows directly from my cases is, that the more pure and uncomplicated the regurgitation, the freer the heart from any other form of disease, the more likely is the individual to be cut off without a moment's warning. Thus, on the one hand, I have watched many a case of aortic regurgitation associated with mitral reflux and aortic stenosis, dilated hypertrophy of the left heart, and eventually tricuspid regurgitation, where death was slowly worked out through the sum-total of subjective miseries and objective changes belonging to the latter form of valvular disease; while, on the other hand, in several of my instances of sudden death, not a single symptom existed to make the victim believe, there was aught amiss within the heart. Here is a case in point. A man, aged about thirty-five, presented himself for insurance. He was a very picture of robust health, and had never had a symptom of disease connected with any organ in his body. Almost purely as matter of form I put the stethoscope to the chest; my attention was at once arrested by a loud murmur, which proved to be basic and diastolic. The arch of the aorta was sound; there was neither hypertrophy nor dilatation of the ventricle; the superficial pulses were markedly visible. To the astonishment of the proposed insurer himself, and to the passing chagrin of the Board, this, to outward seeming, "model life" was refused. The man dropped dead in the street within a fortnight of the refusal.

This paragraph is retained as it stood in the edition of 1863, when written under the impression, the striking fact, it records, in the natural history of aortic reflux, was then, for the first time announced. For, though Corrigan in his original essay actually insists upon the non-occurrence of sudden death in the disease, as affording a guide in its management, while Hope and Bouillaud pass over the matter in silence, and though the connection was certainly not known, or at least acknowledged, in this country in 1855, when I first orally taught it at University College, still, Gendrin had, as early as 1841, established his claim to priority.[*] He was followed by Aran in 1842; possibly anticipated orally by Chomel.

That aortic reflux may at any moment kill instantaneously, and that it kills by syncope, from which nature makes no appreciable effort to rouse the victim, stand, then, as incontestable truths. How is the connection between the two things to be explained? We must bear in mind two propositions:—first, that no other valvular disease exhibits the same inherent disposition to destroy life in a moment,—in truth, no example of really sudden death, through any other uncomplicated valvular disease has ever fallen under my own notice; and, secondly, that, beyond all question, in *some* cases of such death there was no other serious structural change of any kind in the heart or great vessels,—neither cardiac rupture with hæmorrhage into the pericardium; nor aneurism, fatty or cirrhotic metamorphosis of the heart's fibre, nor even dilatation, hypertrophous or attenuated. I cannot, then, accept the proposition of Mauriac:[†] "the true organic cause of sudden death in aortic insufficiency is dilated hypertrophy of the left ventricle complicated with congestion or degeneration." More likely seems his hypothesis that the fatal event comes immediately of embolism of the coronary arteries, paralysing the organ. He properly admits no such obliteration of those vessels has been actually recorded; but was it looked for? Cockle[‡] ascribes the event in great measure to imperfect filling of the coronary arteries, combining with "the disposition to paralysis of the left ventricle from the enormous blood-pressure on its walls." A perusal of both the excellent essays quoted will, I think, impress the reader with a conviction that, no matter how ingenious the reasoning of their authors, the problem remains unsolved at their hands. I regret this the more as I am unable to supply any theory, based on actual observation, capable of explaining the clinical fact. I differ, as has been seen, from Mauriac and Cockle, in believing that instantaneous death may occur not only without, but quite independently of, any cardiac disease beyond the aortic insufficiency.

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[*] Inklings of acquaintance with the tendency of aortic reflux to cause sudden death are to be found in the writings of Morgagni, and of some of the other early masters of observation; but it must be remembered the disease was unknown *clinically* till the day of Corrigan.

[†] De la Mort subite, &c., p. 91, 1860.

[‡] On Insufficiency of the Aortic Valves, &c., pp. 23, 25, 1861.

(*c*) To return to the general question of prognosis in valvular disease. The condition of the heart's substance and the capacity of its cavities are elements of guidance never to be neglected. Deterioration in the nutrition of the ventricular walls, especially with fatty change, carries with it evils of its own, and makes those walls less capable of bearing the extra strain thrown upon them through connected valvular impediment. The cases must be rare, in which fatty metamorphosis lessens the embarrassment of the circulation by weakening ventricular action [558, *b*].

Dilatation of the cavities, if *extreme*, renders any valvular condition more dangerous: and as a rule, hypertrophy, though to a less degree, has the same kind of effect. *Extreme supplemental hypertrophy* of the left ventricle, however, and directly as its purity, may mitigate the effects of aortic constriction; while in the case of aortic reflux, its influence seems practically contradictory. On the one hand, it certainly increases the local evils of the reflux; and yet, on the other, if I may trust my experience, it in some way renders sudden death a less likely contingency [*g*]. I here refer to conditions of hypertrophy unassociated with *notable* dilatation. It must be remembered dilatation, unassisted, is capable, if *extreme*, of giving rise to sudden death.

(*d*) In estimating the prognosis in any particular case, the amount of secondary mischief, pulmonary, systemic, renal or cerebral, affords valuable aid. The "physical signs proper" are always useless, sometimes deceptive, as prognostic guides. No greater error can be committed than that of supposing the danger of valvular diseases, as a class, may be estimated by the amount of murmur, they habitually entail; *exempli gratiâ*, tricuspid regurgitation, which holds so high a place in the scale of fatality, is precisely the defect attended with least constant murmur. Again mitral constriction, constitutionally so grave in its effects, may even in its extremest degrees prove unproductive of murmur. Here is sufficient proof, that implicit faith—nay, any faith—in respect of prognosis, must not be put in the existence of valvular murmurs; and it may be well here, again, to remind the younger reader of what may be fully gathered from the descriptions in the first part of this volume, that there is no direct connection between the amount and danger of disease at an orifice and the intensity of an established murmur; the very *weakness of a murmur may, indeed, be a fatal sign.*

(*e*) But the natural history of valvular disease is probably not one of wholly unmixed evil. There is *à priori* probability, at least, in the view that, where the minute vessels of the brain are positively and gravely diseased—atheromatous, calcified, inelastic, weakened, and fragile—life may be prolonged through the interposition of a constricted state of the mouth of the aorta. So, too, it is conceivable, the lungs (where the disposition to congestion and the actuality of capillary weakness have pre-existed), may be saved from serious textural disease through the instrumentality of tricuspid regurgitation.

(*f*) And, again, it is true, though to all seeming paradoxical, that the sufferer from certain forms of valvular disease, will be placed in a better position, especially as regards probable prolongation of life, by having one or more other forms of disease of the orifices added to that, by which he is already afflicted, than if he retained his original impediment uncomplicated. Take the instance of a sufferer from grave mitral obstruction, who, on the eve of pulmonary apoplexy or œdema, becomes the subject of sequential tricuspid regurgitation,—which, though profoundly mischievous in itself, nevertheless, *pro tempore,* wards off pressing danger by relieving engorgement of the lungs and transferring intra-vascular strain from the pulmonary to the systemic capillaries.

(*g*) The prognosis of aortic reflux is of an exceptional kind. If what has been above stated be correct, no one, in whom unmistakeable evidence of the defect is found, can be considered safe for an hour, though life may be prolonged an indefinite time. The prognosis is very much the same as in a case, where a genuine paroxysm of angina pectoris has occurred [345]. Established tendency to syncope would specially increase the gravity of any particular case. Moderate coexistent dilatation and hypertrophy are useful rather than otherwise; they lessen the evils of regurgitation, the former by allowing a larger quantity of the reflux blood to accumulate in its interior, without too forcible excentric pressure on the wall of the ventricle,—the latter by tending to maintain such power of propulsion as shall prevent systemic stasis.

The greater the reflux through the orifice, the greater the danger; but how can we estimate its amount? Better (but not altogether surely) by the intensity of the specific characters of the pulse, than by those of the murmur. If with a diastolic aortic murmur, the second sound is audible in the carotid, we may conclude, according to Broadbent* and Sibson, that the aortic insufficiency is inconsiderable.

(*h*) Probably no organic diseases present, so frequently as valvular affections, this problem for the observer's solution: how and why is it that a given structural defect can have existed for years without giving rise to serious disturbances, while it at length suddenly produces disordered dynamism in various parts of the frame threatening to destroy life at an early period? Abrupt increase of the valvular mischief sometimes acts as " the last straw that breaks the camel's back,"—sometimes failing innervation of the heart,—sometimes change in the relative amounts of valvular obstruction and supplemental dilatation and hypertrophy,—sometimes intercurrent congestions and inflammations,—but, whatever be the influence destroying the balance of power in the organ to maintain its own against the valvular difficulty, it oftentimes baffles discovery.

(*i*) May the diathetic conditions, under which a given valvular affection has occurred, help in estimating the prognosis? Latham and Broadbent (*loc. cit.*) insist strongly on the more favourable chances, when the mischief acknowledges rheumatic, rather than degenerative, origin. In the former case Broadbent argues the morbid state becomes stationary; in the latter continues progressive. But it cannot be questioned that inflammatory exudation-products never lose their inherent tendency to contract: the longer the time therefore, the greater the contraction and the greater the valvular defect. But admitting the main distinction to be clinically true, the real explanation is probably to be found in the constitutional states severally of rheumatic and degenerative patients. Syphilitic origin may be of favourable augury.

(*k*) Again, aortic disease is graver than mitral, in that it is more likely than the latter to become associated with angina pectoris [341].

(*l*) Finally, in estimating the probable relative fatality of different valvular affections, their several amounts of tendency to produce embolism must not be forgotten. Now a valve-disease may be specially dangerous under this aspect: (1) because detachable vegetations form with excess of frequency at the connected orifice; (2) because, being formed, they are, as matter of fact, oftener detached therefrom; or, (3) because distal impaction is specially dangerous as the sequence of detachment from one valve rather than another. Now vegetations form so rarely at the tricuspid and pulmonary orifices, that from the embolic point of view diseases of the valves of the left side must be notably graver than those of the right. Then, as concerns the mitral and aortic valves, whatever be the relative frequency with which fragile thrombi, exudation-patches or vegetations form upon them [408], detachment has been observed most frequently from the mitral: and of the gravity of cerebral embolism originating from the mitral orifice [608, *b*] no doubt can be entertained. What is the relative measure of solubility in the blood-current of vegetations detached from these two orifices respectively? Probably there is no difference in this respect.

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* On Prognosis in Heart Disease, Brit. Med. Journ. June 9, 1866.

The whole question is a different one from that of the washing onwards of thrombal or exudative particles from the interior of the heart's cavities themselves.

TREATMENT OF VALVULAR DISEASE.

633*. The textural and, consequently, the mechanical condition of imperfect valves (whether their defect be congenital or acquired, traumatic or the result of disease, whether it be of inflammatory, ulcerative, atrophous, cirrhotic, fatty, or calcified nature), lies without the pale of direct treatment. In the present state of knowledge this clinical truth is too self-evident to require comment.

634*. The province of art, then, in regard of valvular diseases, is limited to preventing their occurrence if possible, and to mitigating their effects where developed.

635*. We may, looking at the question from one point of view, lessen the frequency of traumatic mischief to valves by emphatically warning youths—especially of weakly frames—against excess in athletic sports (p. 392). We may sometimes prevent endocarditic disease of valves by placing rheumatically disposed patients under climatic influences favourably influencing the rheumatic diathesis; and also by treating with decision, as far as our present lights guide us, already developed rheumatism.

636*. Once valvular diseases are established, although they be anatomically incurable, their worst functional effects may be long, in some cases indefinitely, averted, by measures accordant with a common-sense view of their nature and ratified by experience.

Whatever be the valve implicated, the treatment is directed, not towards its own disease, but towards:—

(*a*) The condition of the heart's substance;
(*b*) The state of the capillary systems;
(*c*) The general health;
(*d*) Accidental symptoms and occurrences.

(*a*) *Condition of the heart's substance.*—Every day's experience shows that the great desideratum, in regard of the capacity of the cavities and thickness of the valves, is that both shall maintain such proportions as to balance the valvular difficulty with all attainable accuracy. The practical aim, then, must be to prevent force and capacity passing beyond, or to ensure their reaching, the just supplemental point: the control of excess-strength, or the support of undue weakness, becomes the physician's task. Where perfect balance of force exists, symptoms may be wholly wanting: now this perfect compensation may occur spontaneously, or be artifically either actually effected, or, at the least, sensibly promoted.

Practically, therefore, as concerns the heart itself, the treatment of valvular disease is resolved into managing force and capacity, as far as existing knowledge enables us to modify one or the other. First, we may be called on to control hypertrophy with dilatation, on the principles already laid down,—that form of enlargement occupying about the whole of this portion of the therapeutic ground, so rarely is pure or attenuated dilatation associated with valvular defect. Secondly, our province may be simply that of endeavouring to increase the heart's systolic tone.

But hypertrophy, as we have just seen, may or may not be an evil; how are we to determine, whether it be noxious or the reverse in any particular case? Not by ascertaining its amount; not by theorizing on the nature of the valve-defect and the relationship of the hypertrophy thereto; but by estimating the state of the various capillary systems, and by watching for symptoms indicative of stasis and obstruction within them.

Assuming that the evidence shows the heart-force is in excess, the nourishment of the organ may be controlled by occasional leeching over, or even cupping in the vicinity of, the cardiac region. Free blood-letting is unquestionably improper; it cannot remove hypertrophous substance a whit more than the connected valve-affection, and may induce anæmia, venous stagnation, and early dropsy; besides in some valvular defects, above all aortic insufficiency, any risk of syncope is especially to be avoided. Aconite, in doses of two minims, and upwards, of the B. P., may be given two or three times a day.

As regards increase of tone, this is to be effected by the administration of quinine and iron, of the mineral acids, by the cautious use of nux vomica and strychnia [274], and, under certain circumstances, of digitalis.

(*b*) *State of the capillary systems.*—Of the value of *diuretics*, when dropsy has appeared, no doubt can be entertained: but long before matters have proceeded to this issue, medicines of the class seem useful, by lessening venous tension, by diminishing the quantity, without impoverishing the quality, of the circulating fluid, and so lessening the tendency to clogging of the intra-cardiac circulation and the proneness to palpitation. The acetate, nitrate, iodide, and bitartrate of potass (the latter in two-drachm doses largely diluted), nitric ether, compound tincture of iodine, the infusion and spirits of juniper, the decoction of chimaphila, and other agents of the class may be variously combined and successively employed. The action of these diuretics is facilitated by occasional small doses, at bed-time, of blue pill and squill. When the kidneys are much congested, removal of that congestion must be effected, wholly or partially, before the organs will act. Cupping to a small extent, or dry-cupping, in the renal regions sometimes exercises a remarkable effect in facilitating the action of diuretics by removing congestion: in the same way is to be explained the favourable influence of mustard poultices and even *blisters* to the loins. Urine, more or less impregnated with albumen, previous to these measures, sometimes becomes perfectly free, to ordinary tests, of that principle after their employment.

Where the lungs tend to engorgement, espécally in mitral disease, *expectorants*, either ipecacuanha, lobelia, stramonium, or squill and ammoniacum or senega, according to the active or passive character of the symptoms, become necessary. Dry cupping of the chest, sinapisms, and blisters materially relieve such engorgement.

The treatment of dropsy has already been considered [513].

(*c*) *The general health.*—Regularity of alvine discharge, in all diseases important, is particularly essential in these cases, to prevent engorgement of the liver, and obviate the necessity for effort in the act of defecation. Continued purgation is, however, to be avoided prior to the occurrence of dropsy.

If at any time the stomach be loaded, and its replete state excite palpitation, an *emetic* seems clearly indicated: the sulphate of zinc is the fittest. But where the circulation is very much embarrassed, emetics sometimes increase considerably that embarrassment, and unhappily it is far from easy to fix beforehand the probable influence of an emetic in any particular case. If there be doubt, it is better to refrain, and allow the stomach to free itself gradually in the natural manner. Hope was of opinion, and with much *à priori* reason in his favour, that a state of prolonged nausea, by causing languor of the circulation, promoted the formation of fibrinous coagula within the heart.

Antacids, carminatives, and light bitters, and other stomachic medicines, relieve the gastric discomfort so common in the victims of valvular disease; but the latter must not be permitted to an extent to stimulate the appetite to any great amount.

The propriety of administering *tonics* (strychnia, quinine, &c.) will, in the main, turn on the condition of the heart's substance,—whether dilated or hypertrophous. Theoretically, invigorating tonics are more advisable in aortic and in mitral constriction than in aortic and in mitral regurgitation. Anæmia peremptorily calls for iron.

In regard of diet, no constantly applicable rules can be laid down, except that moderation is important both in solids and, especially, in fluids.

Exercise should never be pushed to fatigue, and laborious efforts of all kinds systematically avoided.

When the dyspnœa is of serious character, the toil of mounting a flight or two of stairs, especially if the patient be obese, is more than he dares encounter: his instinct apprises him of the imminent risk of the attempt. Under these circumstances the common plan is to recommend the patient to occupy the ground floor,—a mode of living having its own hygienic disadvantages, unless in houses constructed on an unusually large and commodious scale. W. B. Hutchinson, in a case of this kind, succeeded in enabling the sufferer to use a sleeping-room at the top of the house, much to the improvement of his general health, by having him raised in a chair suspended by solid pulleys to the ceiling of the staircase,—something on the plan of an ordinary hotel "lift."

The use of tobacco in any form or amount is most strongly to be deprecated [314, *b*].

(*d*) *Accidental symptoms and occurrences.—Anti-spasmodics*, chloric ether, ammonia, &c., are necessary in paroxysms of dyspnœa. *Opiates* cannot be dispensed with at night in advanced cases; but where the valvular obstruction is considerable and the heart weak, caution is required in their exhibition. This is equally true of *chloral*, which should be most sparingly used, where the heart is feeble and obstructed [637].

Issues or *setons* to the præcordial region sometimes relieve local pain and discomfort; they are otherwise valueless. At the very earliest period of chronic inflammatory changes in the valves, it is possible that ioduretted frictions may promote absorptive action.

The treatment of palpitation [305], of anginal feelings [347], and of tendency to coagulation within the right cavities [419, *b*], is considered in the paragraphs indicated.

637*. The applicability of digitalis and of drugs, possessing a similar kind of influence on the heart, in the management of valvular disease, may be gathered from what has been said in a former place [305, *a*]. Plainly, extra caution is required in the use of such agents, where the blood-flow within the cavities is impeded. Theoretically, if the beat be notably slackened, where embarrassment of the intra-cardiac circulation already exists in consequence of valvular difficulty, that embarrassment must be increased. And, as matter of experience, there can, I think, be no doubt that evil of this form is occasionally produced by the hap-hazard and continued administration of digitalis,—actually eventuating sometimes in intra-cardiac thrombosis. It is true various cardiac paræsthesiæ, and even downright pain, are occasionally relieved in so remarkable a manner by the drug, as to justify its well-known epithet—"the opiate of the heart." But it seems a mistake to purchase the relief of uneasiness and pain, at the risk of shortening existence by yet further increasing the difficulty of circulation through the organ.

On the other hand, where weak, *irregular*, and over-frequent action, accompanied, or not, with cardiac paræsthesiæ and distress is the dominant state,—and whether this condition of things, culminating in the *asystolism* of Beau, be a sudden, or a gradually induced, disturbance in the heart's dynamism,—digitalis in moderate doses will *sometimes* slacken and regularise the beat and invigorate its tone. This result (even though, as is the fact, it is not invariable), is the more remarkable, as the very same perversions will be induced in a comparatively quiet organ by over-doses of the drug. The determination of the dose then becomes all-important. Twenty minims of the tincture may with propriety be administered in a diffusible stimulant draught during the gravest attacks of quasi-syncopal palpitation, and even where the dominant valvular defect is aortic reflux. But I hasten to add, that I have actually seen this only in cases, where other cardiac mischiefs were associated with the aortic, and where, consequently, as I have been led to judge, the danger of instantaneous death is less than in simple insufficiency [662, *b*]. On the other hand, there is no doubt digitalis is more useful, as a rule, where the defect is mitral than aortic.

Digitalis, as Withering long ago proved, may remove cardiac dropsy again and again, relieving or removing at the same time, pulmonary, hepatic, and renal obstructions; but a time at last comes, when the disease triumphs, and digitalis not only fails to relieve, but distinctly aggravates, the symptoms.

If digitalis be indicated as an occasional medicine, the tincture is probably the best form; if a course be required, the infusion of the fresh leaves is undoubtedly preferable,—the diuretic action, commonly induced, contributing to ward off ill effects.

Of digitaline (whether by the mouth or endermically) my experience is limited,—the chief value of the alkaloid seems to consist in the accuracy with which the amount of really effective principle may be dosed. I confess I have failed to observe the remarkably beneficial effects from its administration, that appear to have impressed some persons.

Chloral, if cautiously given in small doses, acts well as a sedative and hypnotic in cases of valvular disease with cardiac distress and palpitation. But there is extreme risk in administering a large dose (say, thirty to forty grains) in any case, where the idiosyncracy of the individual in regard of this powerful agent is unknown [305, *a*]. Whether there be any connection between the syncopal effects induced by large doses and the distinct fall in temperature (sometimes 2 or 3 degrees Fahr.) they produce, I am unable to say; the connection seems a very likely one. Digitalis, also, though to a less degree, lowers temperature.

§ IX.—CARDIAC BREATHLESSNESS.

638.* Defect in the respiratory act depending on the heart—the absence of pulmonary and hæmic conditions capable of causing dyspnœa being matter of demonstration—is of two main species: it may be dyspnœal or apnœal.

A.—DYSPNŒAL SPECIES.

639*. There are several different varieties of cardiac dyspnœa, clinically very distinct from each other, however closely they be related in physiological mechanism.

640*. *First Form.*—The simplest form of cardiac breathlessness is characterized by tendency to sigh—the patient being conscious or unconscious of the act. In fatty metamorphosis of the heart the habit of non-emotional sighing sometimes proves one of the earliest symptoms.

641*. *Second Form.*—Next appears the peculiar hurried and agitated breathing which occasionally seizes cardiac sufferers while in a state of perfect repose, while sitting or even lying down, and calmly conversing, not the least movement of body having taken place, nor any emotional disturbance having occurred to excite the heart or disturb the breathing rhythm. Attacks of this kind are generally short-lived, and disappear as suddenly and as inexplicably as they came. The respiration, while they last, is loud and panting; the frequency of the act may suddenly rise from 20 to 40 per minute, and the disturbance may be accompanied with very sensible, though never severe, cardiac distress.

641†. *Third Form.*—Breathlessness under exertion is generally first felt in the attempt to get up an ascent in walking, or in going up-stairs. Distress of breathing may eventually occur in walking on a level plain, or on going down a gentle declivity; some persons are able to ride on horseback without annoyance, long after they have lost the power of walking comfortably on level ground. Eventually the least movement—that of turning on a couch, or even changing the posture in sitting—will suffice to induce passing disturbance of the kind.

642*. *Fourth Form.*—But a graver condition of things is where abiding dyspnœa exists (probably in association with pulmonary congestion), combined with occasional paroxysms of intense struggle for breath, where all the thoughts and acts of the sufferer seem absorbed in and directed to the one effort—to breathe. In this pitiable condition sleep, except in the shortest snatches, becomes an impossibility; while hopeless attempts to find a posture, in which chest-motion may be best aided, fill the weary hours. The attitudes occasionally assumed in the agony of the dyspnœa are most singular.

643*. *Treatment.*—These paroxysms of dyspnœa may be relieved by hydrocyanic acid, cannabis indica, æther, and liquor opii sedativus, and the æthereal tincture of lobelia inflata; by dry-cupping, or the application of three or four leeches to the præcordial region, especially if there be palpitation; or by the use of Junod's exhausting apparatus.

Some sufferers of this class obtain sleep in the sitting attitude, with the head bent forwards and artificially supported. I have succeeded often in giving relief of the kind by means of a broad belt slung from the roof of the bed or the ceiling, on which the patient might rest his forehead. But the necessity for this simple contrivance is practically superseded by Dobell's "heart bed," which fulfils not only the present, but many other, indications in managing the posture of cardiac sufferers.*

B.—APNŒAL SPECIES.

644*. Two forms of failure in the breathing act may be referred to this head.

655*. *First Form.*—This singular perversion of breathing (first spoken of by Cheyne, of Dublin, in 1816) may be described as follows. A series of inspirations takes place of gradually increasing force, at a rate of speed, slightly accelerating as they proceed, until a certain maximum is reached. An ascending series being thus completed, a descending follows of force gradually decreasing, until respiratory sound can scarcely be caught through the stethoscope. This immediately precedes a period of apnœa,—neither chest motion nor breathing-sound being perceptible. So complete, indeed, is the inaction of the lungs, that were not the pulse felt, the patient might be supposed dead, until a low inspiration marks the commencement of a new *crescendo* and *diminuendo* series of breathing acts. Beyond these changes in intensity and rhythm, there is nothing unusual in the pulmonary physical signs,—no sibilus or other indication of spasm of the bronchia. Neither is there, as far as I have seen, any evidence of subjective respiratory distress, or, indeed, of suffering of any kind during the suspension of breathing. But freedom from suffering does not, however, always appear to hold during the entire paroxysm. Stokes speaks of "every muscle of inspiration being thrown into the most violent action, accompanied often by a loud moan from the patient."

In Cheyne's case, each revolution in the state of breathing occupied about a minute; the apnœa a quarter of a minute; the number of respirations in a minute equalling about thirty. In a case observed by myself some while since,* the *apnœa lasted thirty seconds on each occasion*—the recurrence of the fits of perverted rhythm being irregular. The phenomenon may continue for months in a minor degree, and may occur during sleep: at least the testimony of the patient's wife, in the case just referred to, was very positive on these points.

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* Vide description by H. Bigg in Brit. Med. Journal, Feb., 1872.

656*. This singular condition of breathing is of extreme rarity. In the course of many years I have met with but three examples in which the apnœa was as complete as in the instance just referred to.

657*. This form of apnœa was long held to be peculiar to, and distinctive of, fatty metamorphosis; and, as far as my experience goes, its *perfect* development belongs to that disease alone. But in lesser degrees (as was first noticed in print by Seaton Reid†) this strange perversion may accompany many cardiac affections of enfeebling character,—it has no specific connection with fatty disorganisation, weakness being probably, as far as the heart's substance is concerned, the only necessary element [559, *g*].

Admitting this cardiac feebleness as the ground-work, it must be remembered any statical weakness of texture is an *abiding* condition, and that some dynamic disturbance must be sought for in explanation of the actual and only *occasional* paroxysm. What is the nature of that superadded disturbance? Can it be gradually increasing weakness, eventually carried to quasi-paralysis, of the *right* ventricle,—the blood stagnating in the lungs until the instinctive craving for oxygen again becomes urgent enough to set the apparatus in feeble motion? But the pulse at the wrist holds on unchanged during the paroxysm; why should the *right* ventricle fail in action, while the *left* thus proves itself to be vigorous? Besides, in the case above referred to, jugular pulsation continued, and the cardiac sounds at the pulmonary cartilage maintained their fulness, during the apnœa. Neither are there any conditions of brain present (unless such pre-existed), during the entire fit of perverted rhythm, pointing to stagnation within the cranium. It probably lies beyond the powers of existing physiology to explain satisfactorily the mechanism of this apnœa; but, guided by the facts just referred to, I cannot avoid adhering to the opinion formerly expressed, that the *proximate* cause lies in a failure of the special nervous excitant of the respiratory act,—in anæsthesia either of the vagus or of the medulla oblongata itself: this anæsthesia would in turn be of course dependent on the dwindled and sluggish blood-current propelled by the languidly contracting heart.* The perversion of rhythm, though differing in character from, is probably allied in mechanism to, the state I have elsewhere described as "nervous apnœa."

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* In conjunction with King, Elliott, and McMeikan of Stratford.

† Dub. Hosp. Gaz., 1860.

658*. *Second Form.*—In some rare examples of cardiac disease—so rare that the peculiarity is little known and less understood—there is a tendency to complete stoppage of breathing, the moment the patient, falling into the oblivion of sleep, fails to excite the respiration-act by volition. This is a condition of extreme danger,—and one which happily never occurs (at least it has never occurred under my observation), except where various other concomitant symptoms point to death as imminent and inevitable.

659*. Is there anything to be done to lessen the danger attending fitful dozes in these deplorable cases? I think I am justified in saying, that the tendency to arrest of respiration may to a certain extent be averted by the use of a gentle electro-galvanic current playing from the nucha to the epigastrium. I have certainly tried this with some benefit.

§ X.—ANEURISM OF THE HEART AND VALVES.

I.—Heart.

660*. *Anatomical characters* (*a*).—Aneurism of the heart occurs in two forms, corresponding to the fusiform dilated aneurism, and the lateral simple sacculated aneurism, of arteries (*vide* Diagram, figs. 1, 3 [775] : that is, a general and tolerably equable dilatation of a portion of the wall of a ventricle exists, or a pouched fulness rises abruptly, with or without constricted orifice, from the ventricle. Of either kind, aneurism is almost peculiar to the left ventricle; of seventy-four cases, collected by Thurnam, fifty-eight were examples of the disease in the left ventricle,—while in no single instance was the right affected. The compound sacculated aneurism of arteries, with injury to their walls (Diagram), fig. 4, [775], is also imitated by the heart, when destruction of the endocardium and, more or less extensively, of the nearest strata of muscular fibres, precedes the pouching process.

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* Disease of Heart, 3rd ed., p. 345, 1862. The same view has recently been advocated by Fraentzel (Berlin. Klin. Wochenschrift, 1869), who, like Traube, believes he has observed the phenomenon in cerebral affections and uræmic coma, the heart being free from disease (Syd. Soc. Year Book, 1869—70).

(*b*) Sacs of recent formation are generally provided with a wide orifice; as the sac ages, the opening relatively loses in diameter, while its lips grow prominent, well-defined, and fibroid.

(*c*) There may be little or no bulging on the pericardial surface of the ventricle, indicating the seat of the disease; or an elevated prominence, varying in size from that of a nut to that of well-nigh the heart itself, may exist. And when, as is not very uncommon, the septum is the seat of the aneurism, the sac need not in the least alter the external form of the heart.*

(*d*) Aneurismal sacs have been found empty, or have contained coagula, either laminated or amorphous,—all three states seem to occur with about equal frequency.

(*e*) In the vast majority of cases a single sac only has existed; but not a few examples of two, and a rare one of three or even four coexisting sacs, are to be found recorded in periodical works. When numerous, the sacs may coalesce.

(*f*) The reality of processes of curative tendency occurring in cardiac aneurism has now and then been substantiated; indeed, actual cure has occasionally been accomplished by the complete obliteration of the sac with fibrine.

661*. *Course.*—In the majority of cases aneurism forms slowly, dependent, as it is, for existence on chronic changes in the substance of the heart; occasionally, however, the formation proves an acute process, induced by ulceration or rupture of the endocardium and contiguous fibres. Once produced, the structure, forming the wall of the sac, may undergo secondary changes in the shape of cirrhotic, fibroid, cartilaginiform, or ossiform infiltration.

662*. *Causes.*—Greatly more frequent (as 3 to 1) in males than females—hitherto most commonly observed between the ages of twenty and thirty and in very advanced life, though few ages are actually exempt from the possibility of its occurrence,—having the ground-work laid for its formation in inflammation, fatty change, simple softening, cirrhotic [590] or pseudo-fibrous infiltration of the heart's substance,—aneurism has been immediately traced, in a certain number of instances, to external injury, violent efforts, forced retention of the breath, and similar agencies, suddenly throwing an intense strain on the walls of the left ventricle.†

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* Univ. Coll. Museum, No. 4592; sac protruding through septum below aortic valves, communicating with the left, and bulging into the right, ventricle.

† Prolonged stage-effort has been assigned as the cause of the cardiac aneurism of

In the majority of cases its clinical, as well as its anatomical, origin is slow and insidious—in fact, latent.

663. *Symptoms.*—Under all circumstances, whether the production of the aneurismal pouching be an abrupt and rapid, or a deliberate and slow process, the symptoms are obscure.

(*a*) Where the onset has been presumedly sudden, the heart's texture lending itself through local structural weakness to quasi-instantaneous bulging, the occurrence has beyond doubt been attended with a certain number of grave cardiac disturbances,—such as severe præcordial pain, orthopnœa, alarm and general agitation, dread of dissolution, syncopal tendency and frequent, small, irregular, languid pulse. But obviously there is nothing distinctive in these symptoms: they indicate that the heart has received a deep shock of some kind, and nothing more.

(*b*) On the other hand, where the disease is of slow origin and course, its symptoms are non-specific, and do not seem to become serious, unless itself is carried to a great amount, or unless dilatation, with more or less hypertrophy, is added. The effects of such dilatation are then developed—systemic stagnation and its attendant evils in more or less complete array. At all events, it will be admitted that past records do not supply the materials of a clinical history of aneurism, clearly distinct from that of affections of the ventricles and of the orifices, however complete an anatomical one they have been made to yield by the zeal of Thurnam. Every known symptom of cardiac disease has been noted, it is true, in these cases; but, as there either were positively, or may possibly have been, other morbid states present (I refer to narratives which make no affirmation as to the absence of such states), capable of causing the symptoms in question, it would be perfectly unjustifiable to ascribe them to aneurism.

664. *Physical signs.*—And there is as great a dearth of knowledge of positive physical signs.

(*a*) The position of the heart's maximum impulse may perhaps be transferred from the apex to the base, where a sacculated aneurism springs from the neighbourhood of the base of the ventricle; probably, if the sac were prominent, the action of the heart would be attended with pericardial rub, not only systolic, but diastolic. Actual tendency to present externally, after adhesion of the sac to the chest-wall, has been met with; the pecu-

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the great French tragedian, Talma; but as he had long laboured under stricture of the rectum, it would appear the perpetual strain in defecation must have played the part of a much more active cause.

liarity does not appear to have led to successful diagnosis,—though it is conceivable the nature of the affection might at least be surmised through its existence.

(*b*) Systolic murmur of blowing quality, of maximum force at the left apex, has been observed, undistinguishable from the murmur of mitral regurgitation, except perhaps by its inaudibleness in the left vertebral groove [109, Table]. Though seemingly probable, *à priori*, that a double murmur might be produced by the ingress and egress of blood from the sac, where the orifice of this was narrow, experience shows that the diastolic portion may be completely wanting.*

(*c*) The signs of dilated hypertrophy frequently, of valvular disease sometimes, are coincidentally met with.

665. *Mode of death.*—Death may occur suddenly from rupture of the sac into the pericardium, or through an adherent pericardium, into the pleura: in the majority of instances the patient has been slowly worn out with the symptoms of cardiac dilatation.

666. *Treatment.*—There is no special treatment for this affection: the symptoms and signs guide the physician to the adoption of the measures best adapted for hypertrophy or dilatation: the effects of systemic stagnation are to be averted or removed by the plans already described.

II.—VALVES.

667. Aneurism, pouching, or sacculation has been observed in the mitral, tricuspid and aortic valves,—habitually, as might be expected, with the concavity of the sac looking in the direction of regurgitation. Doubtless the local distension is produced by the force of the impelled column of blood, acting on textures weakened in resistent power by chronic impairment of nutrition. Generally very small, the sac may equal a hazel-nut in size; it may undergo perforation.

In a specimen of sacculation of the long tongue of the mitral valve now before me,† the sac bulges, contrary to the rule, in the direction of the ventricle. It might lodge a small pea in its interior; and is much thinned, well nigh perforated, at the apex. It is difficult enough to understand how undue strain could be exercised on the mitral valve in this direction; and the mechanism of aneurism thus occurring, as it were, with the blood-stream, is obscure enough.

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* *E. g.*, In case V. of H. Douglas's Collection of Cases of Heart-disease, Edin. Monthly Journal, 1850.

† Univ. Coll. Museum, No. 4166.

668. The clinical history of valvular aneurism is as yet a blank.

§ XI.—RUPTURE AND PERFORATION OF THE HEART.

I.—COMPLETE RUPTURE.

669. *Seat.*—Complete rupture of the heart's substance into the pericardium, from all causes, spontaneous and other, indiscriminately, takes place with much greater frequency in the left ventricle than in any other part of the organ:* but, if cases originating in external violence alone be considered, the right ventricle has, according to existing records, suffered more frequently than the left, in the proportion, according to Ollivier, of eight to three.

670. *Anatomical character.*—Variable in size and form, sometimes smooth, sometimes ragged at the edges, the inner and outer openings—that is, the pericardial and the endocardial—may correspond or not; in the latter case, a sort of sinus exists in the wall of the ventricle, connecting the two. There is usually a single rupture only, but so many as five have been seen; sometimes a single opening on the inner surface of the ventricle communicates with two or more on its pericardial aspect. The strata of muscular tissue next the endocardium, again, may be pretty extensively destroyed, while a tiny perforation only in the pericardium can be discovered. The fissure runs very nearly three times as often parallel to, as at right angles with, the main fasciculi of the heart's fibres.

671. *Course.*—Although there is every reason to believe that, in the great majority of cases, non-traumatic rupture of the heart is perfectly sudden in accomplishment, it is clearly a gradual process in some rare cases; for instance, where hæmorrhagic softening of tissue has led to its occurrence. Under all circumstances, the final breakage of the last few muscular fibres and outer serous membrane, that separate the pericardial and endocardial cavities, takes place instantaneously.

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* In 52 instances of rupture collected by Gluge (Path. Anat.) the left ventricle was the seat of the rupture in 37 cases; the right ventricle in 8; both ventricles in 2; the right auricle in 2; the left auricle in 3.

672. *Causes.*—Considerably more frequent in males than females, in the ratio of 36 to 16, rupture of the heart is favoured by advanced age: it becomes comparatively frequent after the fiftieth, still more so after the sixtieth year. Immediately induced by effort of some kind, by fits of passion, by great and abrupt thoracic congestion, as through sudden immersion in the cold bath, by blows and other injuries to the præcordial region, the way is paved for its occurrence by various textural changes of the heart's substance or aorta;—as fatty accumulation under the pericardium, combined with muscular softness; intra-sarcolemmous fatty necrosis; softening of undetermined kinds; dilatation with attenuation; local suppuration, ulceration, possibly gangrene; hypertrophy, with, probably, fatty change superadded; hydatids; cirrhosis or calcification of the ruptured tissue; aneurism of the left ventricle; local destruction of the endocardium; apoplexy of the heart (the question may, however, be fairly raised, whether in some of the cases where blood-infiltration and rupture have been found together, the former may not have depended on gradual advance of the latter); coarctation of the arch of the aorta; and, as in the case of George I., aneurism of that vessel. In all probability, though old narratives say the contrary, the heart's texture is never perfectly sound; even where the rupture had been caused by external violence, some alteration of tissue has been generally found by recent observers.

673. *Symptoms.*—(*a*) In the majority of cases, rupture of the heart, if actually complete, at all extensive, and instantaneous, kills instantaneously. The hand is suddenly carried to the front of the chest; a piercing shriek uttered; some convulsive twitches occur, and the patient expires: or sudden loss of consciousness, from which recovery never takes place, marks the event.

(*b*) There is a second class of cases, in which, from the plugging of the fissure by coagula, the amount of extravasation of blood into the pericardium is insufficient to stop the heart's action at once. Under these circumstances, a patient has been known to survive fourteen hours, with pallor, cardiac anguish, clammy sweats, coldness of the surface, feeble fluttering pulse, and sighing respiration; eventually going off quietly in a state of coma.

The fact of death occurring so slowly suggests the question, whether the permanent closure of a fissured opening does not fall within the range of the possible; and certainly one case of sudden death from rupture has been recorded, in which a former rupture was discovered, firmly filled by a fibrinous coagulum adherent to the wall of the heart. I have before me a preparation, in which an attempt at plastic closure is shown. Towards the apex of the right ventricle lies a fissure about five-eighths of an inch long; at the upper angle appears a minute aperture leading into the ventricle; while the remainder is filled with lymph, adventitious (?) muscular fibre, and wavy fibres with round particles and granules in their interstices (Univ. Coll. Mus., No. 4077). The clinical conditions, observed at the close of life in this case, may advantageously be placed side by side with this brief notice of the state of the heart.

"William Hutchinson, ætat. 48, presenting all the signs and symptoms of general dilatation, with hypertrophy, of the heart (the organ was found after death to weigh twenty-two ounces), mitral and tricuspid regurgitation, enlarged liver, pulmonary apoplexy, and anasarca, had slightly improved in the majority of his symptoms during three weeks' stay in the hospital, when a change for the worse, attracting the immediate attention of the nurses, occurred on the morning of the 25th August: the countenance had become of pale yellowish earthy aspect; the conjunctivæ bloodless; the face and thorax covered with cold sweat; the respiration laboured and frequent; the pulse, weaker and smaller than previously, unaccountable from frequency; feet considerably œdematous and cold; no pain complained of; restlessness intense,—change of posture almost every minute; tongue particularly clean; bowels naturally open; during the night of the 25th, insomnia, constant restlessness; dressed himself and sat on side of bed greater part of the night, wishing to go home. August 26th. In the morning: does not know his children; body exhales an earthy, and somewhat putrescent odour; extremities cold; pulse almost imperceptible, unaccountable from frequency; respiration more difficult and frequent than yesterday; cold sweats: so late as 3.30 p.m. of this day he got out of bed to sit up; passed urine under him; at 5.0 p.m. hacking cough set in; shortly after, the respiration became infrequent, occurring at such rare intervals that each breath seemed to be his last. . . . Sank at 5.59 p.m., without any change of symptom. On examination (made, in my absence, by Dr. Hare) about two ounces of dark blood, partly coagulated, partly fluid, were found on the surface of the heart and root of the aorta; after careful search, a minute aperture, barely admitting an ordinary sized pin, was detected close to the apex of the right ventricle, and traced to communicate with the interior of that cavity in an oblique direction downwards, and to the left. (U. C. H., Males, vol. viii., pp. 334—354.)

The two conditions of the blood in the pericardium (coagulated and fluid) point to two distinct acts of hæmorrhage; the one coincident with the first and nearly closed rupture, the other with the final perforation. The probability is, the first rupture was effected during the night of the 24th, or the morning of the 25th; and that consequently the patient survived the occurrence thirty hours on the lowest computation.

(*c*) A third class of cases exists, in which sudden cardiac anguish, attended with a sense of constriction, extreme dyspnœa, pain extending from the præcordial region to the left shoulder, coldness of the surface, giddiness and faintness, cramps, and small irregular pulse, is observed,—the whole series undergoing remission, nay, actually, it is said, totally disappearing temporarily, again to return with greater intensity than before, and close in death. In instances of this kind, in all probability, the heart's fibres have given way in successive layers.

674. *Treatment.*—The greater number of cases of at all extensive rupture have terminated fatally, before medical attendance has been procured. Should the patient be still living, when first seen, such treatment should be employed as a common-sense view of the symptoms would suggest; for it is more than probable that the precise nature of what has occurred will rarely, if ever, be diagnosticated with surety. Theoretically, the clear indication is to maintain the circulation in movement, with as little work on the part of the heart as possible; the head should be placed low, sedatives and slight stimulants administered, the surface kept artificially warm, and the very slightest movement of the body, as far as possible, prevented,—but the perpetual restlessness observed in the case above narrated, shows how unlikely we are to secure this theoretical advantage.

In instances of slow perforation, of which an illustration is seen in that case, the nature of the occurrence lies, in the first place, beyond the reach of sure diagnosis. In an individual, dying more or less rapidly of profound organic disease of the heart, the increased uneasiness and increased disturbance of the pulse, depending on perforation, might be taken for indications of polypoid formation within the cavities, or rupture of a portion of valve or chorda tendinea. Fortunately, however, the treatment will not err in consequence: in any one of these cases the immediate management is that fitted for nervous shock and cardiac obstruction.

II.—PARTIAL RUPTURE.

675. *Seat.*—Partial ruptures of the heart, or of its valvular apparatuses, are not extremely unfrequent. Rupture of the papillary muscles, permitting the ruptured ends to float, as it were, free in the cavity of the ventricle; rupture of the substance of the mitral or tricuspid valves, or of their tendinous cords; and of the pulmonary and aortic valves,—have all been observed *post mortem*, and their symptoms occasionally noted with more or less precision during life.

676. *Symptoms.*—(*a*) The immediate symptoms of the rupture of a chorda tendinea, especially if endocarditis have preluded the

accident, are not very distinctive. The difficulty of the circulation through the heart, however, is increased; the pulse becomes very irregular, and symptoms, precisely like those of the sudden formation of blood-concretions in the ventricles, make their appearance. The murmurs, obstructive and regurgitant, of the implicated valve ensue; but they may have pre-existed, as consequences of the endocarditis itself, and in that case positive diagnosis is an impossibility.

(*b*) Sudden rupture of any number of chordæ tendineæ, or of a papillary muscle, when, *per contra*, occurring independently of acute disease of the heart, produces very definite symptoms. Cardiac pain and anguish; palpitation irregular in force and rhythm; small, irregular, frequent pulse; syncopal tendency, overwhelming dyspnœa, dread of dissolution, pallor, coldness of surface, jactitation of the limbs,—all this suddenly occurring in connection with a regurgitant systolic apex-murmur, which had been known not to have existed previously, might guide to the diagnosis, but not justify a positive assertion on the subject.

(*c*) In a case of rupture of one of the aortic valves, observed by myself, symptoms, such as those just described, were attended with regurgitant, without obstructive, basic murmur [115]. But it would appear from a very instructive narrative, lately printed by Henry Simpson,* that pain, faintness, and distress may all be deficient at the time of the accident. In this instance, as in that which fell under my notice, there was no obstructive basic murmur: in my patient the rupture was fresh, in Simpson's about fifty-four months old at the time of observation.

677. *Treatment.*—The character of the symptoms points to the propriety of administering stimulants and sedatives: sinapisms or turpentine fomentations to the cardiac region relieve the anguish and constriction felt in that situation.

§ XII.—MALPOSITIONS OF THE HEART.

678. Malpositions of the heart are *congenital* or *acquired.*

679. (A) Of the *congenital* class the extra-thoracic, cephalic, and abdominal ectopiæ are totally without clinical interest. Not so, congenital malposition of the heart in the right half of the thorax; for this being a kind of displacement producible at all periods of extra-uterine life by a variety of diseases, it becomes

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* Brit. Med. Journal, Aug. 15, 1868.

of importance to have a means of positively distinguishing the malposition of congenital origin. Now this means is furnished by the position of the liver and spleen; for, except in very rare instances, when the heart has formed and grown on the wrong side of the spine, the abdominal viscera, also, have been transposed. Where this guide is wanting, the distinction may be very difficult; and the observer is only justified in pronouncing the malposition of the heart to be congenital, after he has succeeded in excluding every possible morbid source of acquired displacement.

680. (B). The diagnosis of *acquired* displacements of the heart is to be made by the position of the apex-beat, and of the impulse of the organ generally, by the altered locality of cardiac percussion-dulness, and by the comparative intensity of the heart's sounds, more especially of the first, at different parts of the chest.

681. *Species.*—The species of acquired displacement, which possess practical importance, are three,—namely, sideward *detrusion* and *traction*, *elevation*, and *procidentia.* To very slight amounts all three are of very frequent occurrence, a necessary result of the looseness of the anatomical connections of the organ [1]. But, even when carried to extremes, these displacements produce little or no subjective disturbance; their clinical interest really depends upon the light thrown by their discovery on the diagnosis of various intrathoracic diseases.

682. (*a*) *Detrusion* may occur towards either side, but is more striking, and hence more readily discovered, in its minor degrees, when occurring towards the right than the left. In the former position the main impulse may be carried nearly as far outwards as the right nipple, between the fifth and seventh ribs. In the latter, the axillary region may pulsate markedly, while movement is scarcely perceptible, or actually null, at the true cardiac region; the apex is commonly raised by the width of an intercostal space above the normal level.

In both cases the base is less displaced than the apex; the organ not only changing its place as a single mass, but undergoing a rotatory movement, of which the base-region is the centre. Hence it is, that the maximum point of the first sound suffers very much greater change of place than that of the second. It is noteworthy enough that the amount of displacement, actually found after death, is habitually less than the state of the physical signs during life has led the observer to anticipate. I have repeatedly noticed this where all necessary precautions (ligature of the trachea, fixing the heart *in situ* by long needles, &c.) had been taken to prevent the positions of the viscera from being notably altered by opening the thoracic and abdominal cavities.

By far the most frequent cause of lateral displacement of the heart is pleuritic effusion: above all, this is the affection which, in actual practice, most commonly carries the organ seriously out of its place. Perforative pneumothorax, with inflammatory liquid effusion, will push the displacement to its extreme possible limit. Simple pneumothorax is so rare an affection in the idiopathic form, and, where perforative, is so rapidly followed by other morbid conditions of the parts concerned, that its detruding influence on the heart is rather matter of theory than of actual observation. Hæmothorax is rarely copious enough for the purpose; but in a case of aneurism of the arch, terminating by sudden death from extravasation of about two quarts of blood into the left pleural sac, the heart was found, *post-mortem*, lying to the right of the middle line.* True hydrothorax being generally double, and consequently exercising about equal pressure on both lateral aspects of the heart, does not displace the organ sidewards. Intrathoracic tumours and aneurisms,† variously placed, occasionally push the heart to the right or to the left. Hypertrophy and emphysema, if limited to, or in great excess in, one lung, drive the organ proportionally to the other side. Pneumonic consolidation, especially on the right side, displaces the heart slightly in the opposite direction, provided the hepatisation be on a massive scale, and especially if it be rapidly effected; but if the lateral displacement be at all considerable—if it equal an inch and a half—the presence of fluid in the pleural sac may, according to my experience, be looked upon as certain. Finally, hernia of the intestines through the diaphragm may be quoted as an infinitely rare cause of this variety of malposition.

(*b*) The heart may be pushed forwards by emphysema; the displacement is with difficulty ascertained, on account of the presence of the thick stratum of distended lung in front. Intrathoracic tumours and aneurisms lying behind the heart push it forwards, the signs in the latter case especially are very peculiar; the simulation of cardiac hypertrophy may be very perfect.

683. *Traction.*—The heart may be drawn, as well as pushed, sidewards from its normal site. In cases of rapid absorption of right pleuritic effusion, the heart is occasionally dragged into the

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* Bell, U. C. H., Males, vol. vii., p. 169.

† To a very remarkable amount in Moriarty, U. C. H., Males, vol. xii., p. 5.

right thorax at the period of absorption, especially if that absorption be very rapidly effected. So, too, it may be displaced by an analogous mechanism in cases of unusually marked diminution of the bulk of the lung, induced by interstitial contraction of pneumonic exudation-matter. This is much better seen, however, in genuine pulmonary cirrhosis. Slight displacement of the sort occurs in simple (but not in emphysematous) atrophy, limited to one lung. I have seen the heart beating in the right thorax, where the sole explanation was to be found in great reduction of bulk of the right lung, through the contraction attendant on tuberculous infiltration, and on lessening area of large excavations.

684. *Elevation* (*a*).—The heart, as a whole, may be raised above its natural level in all varieties of enlargement of the abdominal viscera, and in cases of liquid and gaseous accumulation within the abdomen. The splenic enlargement of leucocythemia may permanently raise the organ an entire interspace and upwards [*]; enlargement of the liver from abscess will also raise the organ somewhat,—a fact which, as I have elsewhere shown, may become very valuable in a case where the diagnosis between hepatic abscess and abscess in the abdominal wall over the liver chances to present unusual difficulties.[†] The heart may be carried very notably above the normal level by the contraction of a tuberculous cavity in the upper part of the left lung; or, to a less degree, through the diminished bulk of the apex, which attends the earlier consolidating and atrophous stages of the disease.

As far as I have seen, ovarian dropsy is the affection in which elevation of the heart reaches its maximum. I have known it impossible to feel distinct impulse lower than the second interspace, though slight fluttering movement might be seen in the third.

(*b*) The heart's apex may be carried upwards and outwards, while the base remains, practically speaking, fixed, in pericarditic effusion. Of this condition enough has already been said [47, *b*].

685. In *procidentia* the heart lies lower than natural; at the same time it generally sways a little to the right. In cases of prolonged debility, especially of adynamic pyrexia, there seems reason to believe that elongation of the great vessels occurs to a slight extent, and entails a proportional fall of the heart. In hypertrophy and dilatation, though the extension of the heart's

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[*] Dujardin, U. C. H., Males, vol. v., p. 192, June, 1850 ; C. Hayes, U. C. F., Males, vol. x., p. 212 ; also Mr. ——, seen with Mr. Rayner, of Kingsland.

[†] Case of Fairbanks, Clin. Lect., Lancet, *loc. cit.*, 1849.

outline downwards mainly depends on the increased bulk of the organ, it seems, especially in the former affection, in some measure traceable to lowering of level dependent on increase of weight. Mediastinal tumours and aneurisms occasionally push the organ more or less notably downwards. But it is as an attendant on emphysema of both lungs, that this form of displacement is most frequent, and of most practical significance; in point of fact, such change of position of the heart is one of the most valuable signs of that pulmonary affection. Similar displacement may be caused by double bronchitis; but the acute inflammation rarely suffices to disturb the position of the organ, unless old-standing emphysema have pre-existed. Double hydrothorax does not push the heart downwards as invariably as might be theoretically expected.

686. *Treatment.*—There is, of course, no direct treatment of displacement of the heart. The organ is only to be restored to its place by the cure of the diseased states that have removed it therefrom.

§ XIII.—MALFORMATIONS OF THE HEART.

687. There are a certain number of malformations of the heart and its appendages which, interesting enough to those who make special pursuits of morbid anatomy and teratology, possess little or no importance in a clinical point of view. We shall scarcely delay with the description of these.

I.—MALFORMATIONS UNATTENDED WITH SYMPTOMS.

688 (*a*). Deficiency of the pericardium, the heart lying mainly in the left thorax, and at somewhat a lower level than natural, has several times been observed.* In no one instance of which I have read the narrative, from the case recorded by Baillie to those of quite recent date, were any symptoms noticed. In some of the subjects a sort of rudimentary serous sac could be traced.

But though symptoms may be wanting, it seems probable enough the physical examination of the heart's motion might disclose something abnormal. There is no present attainable evidence on the matter.

(*b*) Excessive congenital smallness of the heart would be attended with physical signs undistinguishable from those of acquired concentric atrophy [473]. The state is certainly unproductive of local annoyances of any kind; and though it has been seen in connection with epilepsy, the relationship of cause and effect between the two things may reasonably be doubted.*

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* Univ. Coll. Museum, Carswell's Drawings, A., No. 446—here the fibrous layer only wanting.

(*c*) Perversions of shape, at least anatomically curious, have occasionally been noticed. Thus the apex has been found bifid.†

(*d*) The valves are subject to three main forms of congenital imperfection. Deficiency of number; excess of number; malposition, with deficiency of structure, the tongues of the affected valve, largely perforated, lying across the blood-current.

There cannot be a doubt, certain of the conditions, falling under these heads, must be productive of abnormal physical signs; though, what these may precisely be, experience has not yet taught us. Of subjective effects, if such occur, nothing is yet known.

Were physical signs to occur in connection with the malformed semi-lunar or auriculo-ventricular valves, they must of necessity be significant either of obstruction, of reflux, or of both; nor can I imagine the possibility of distinguishing such mechanism from that of the murmurs of acquired disease. Yet there are contingencies under which such power of distinction would be practically useful; as for instance, in individuals labouring under certain diathetic affections (rheumatism, Bright's disease, &c.), prone to engender endocarditis,—and who are found to present a morbid sound bearing all the attributes of a murmur caused by inflammatory changes, but nevertheless, it might be, really resulting from congenital malformation. However, this matter has already been considered in its practical bearings as far as needful [459].

II.—MALFORMATIONS ATTENDED WITH SYMPTOMS.

689. There are, on the other hand, a considerable number of malformations of the heart which, generally speaking, not only prove more or less incompatible with prolonged extra-uterine life, but also, while that life continues, produce more or less grave symptoms. Among these symptoms, though it yields to many others in real importance, a certain blueish or livid discoloration of the skin has always attracted most attention—to such a degree, that this symptom, with its functional accompaniments, has been

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* Nasseau, Arch. Gén. de Médecine, t. viii., p. 603.

† Parise, Bulletin de la Soc. Anat., 1837.

by the nosologists elevated to the rank of a specific disease, under the titles of cyanosis, or morbus cæruleus. And however indefensible scientifically this mode of treating the subject may be, it carries with it so many practical advantages that we will not depart from it here. Let us proceed, then, to review the group of clinical conditions characterising cyanosis.

CYANOSIS.

690. *Symptoms.*—(*a*) The discoloration of the tegumentary membranes, especially that of the skin, may be referred to two main types—blue, and pale claret. As will hereafter appear, this distinction is probably not unimportant. The blue colour ranges between various shades of light or dark leaden, purplish, or almost black; the claret tint may be very light, or of the deepest shade that is ever seen in *post-mortem* vibices, or even in rubeola nigra.

The discoloration may pervade the whole surface, but attains its maximum at the internal canthi, lips, nose, ears, below the eyes, about the genital organs, and at the tips of the fingers and toes. Changeable in intensity from time to time, the tint greatly deepens from palpitation, effort, fits of ill temper, dyspnœa, and, in a word, from all conditions adding any functional obstacle to the abiding statical difficulty of the circulation. So long as respiration remains easy, it is undeniable that in some instances *the tegumentary colour scarcely deviates from the natural hue.* Generally speaking, the colour, of whichever type it be, lightens after death,—a fact, in a certain sense, the counterpart of the change of colour noticed in the dead bodies of people cut off in the algide stage of cholera Asiatica.

(*b*) The ends of the fingers, if life continue for any length of time, grow more or less bulbous; in adults this peculiarity of form is sometimes singularly well-marked;* at the same time the nails become incurvated; and the patients have been found particularly subject to whitlow, and to sluggish cutaneous sores about the mucous orifices.

(*c*) The temperature of the surface and mucous canals, at their outlets, is low; the patient, constantly chilly and sensitive to the least fall in the thermometer, will bear a large fire in the warmest weather, this climate affords.

(*d*) Œdema of the feet sometimes exists.

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* Occasionally pushed to greater extremes than is ever seen even in cases of empyema,—*e. g.*, Univ. Coll. Museum, Drawings, A. No. 711.

(*e*) As a rule the frame is weak, the muscular and adipose systems ill-nourished, sometimes peculiarly wasted. Nevertheless, even where cyanæmia has reached the extremest limit,—where the whole system, practically speaking, derives its sustenance from dark blood, the viscera may be of full size, and in point of colour present no serious difference from healthy organs. At least such I found to be the fact in a case where the aorta rose from the right, and the pulmonary artery from the left, ventricle, while the great veins held their normal relationship to the right and left auricles, the only medium of communication between the two distinct circulations, capable of conveying florid blood to the systemic capillaries, being the ductus arteriosus. And in this case, as the blood in the portion of the aorta lying between the opening of the ductus arteriosus and the heart was wholly venous, and as the coronary arteries rose from the aorta in the usual position close to the sygmoid valves, it follows, the nutrition of the heart, which was hypertrophous, must have been wholly effected by non-oxygenated blood.[*] Possibly the slight degree to which the colour of the viscera differed, in this instance, from that of health may have depended upon their tint, as well as that of the tegumentary membranes, having lightened after death. Louis states that the colour of inflamed organs is the same as in persons with naturally coloured blood.[†]

(*f*) The digestive faculty is habitually feeble; and in a certain number of cases more or less persistent diarrhœa has contributed to enfeeble the system or even destroy life.

(*g*) Though rarely the subjects of serious habitual dyspnœa, cyanotic persons, with the rarest exceptions, suffer from paroxysmal difficulty of breathing—often the result of disturbed circulation, caused by fits of petulance and passion, to which they prove, above the average, prone. Cough, either dry or attended with watery expectoration, probably through œdema of the lungs; frequent attacks of congestive bronchitis, added to, sometimes, the physical signs of emphysema, or of atelectasis, congenital or acquired, or of all three combined; contribute their share to the sum of pulmonary symptoms and morbid states.

(*h*) In many cyanotic cases the heart is habitually quiet enough; but paroxysmal disturbance of its action is common to them all. Palpitation, active, tumultuous, with strong diffused impulse, a pulse ranging from 120 to 160, irregular in force and rhythm, and sometimes almost imperceptible, commonly co-exists with the dyspnœal paroxysms above referred to; syncopal tendency and actual syncope, though rare, do sometimes actually occur; the eyes are prominent, the expression wild, and the arms tossed violently about. The duration of these fits ranges from a few minutes to some hours; and they may be of frequent, daily, nay, almost hourly, occurrence, or re-appear at rather rare intervals. There is no particular pulsation observable in even the most deeply-coloured parts, though the capillary vessels and arterioles are sometimes distinctly enlarged.

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[*] Medico-Chirurg. Transactions, vol. xxv. Nasse affirms the spleen, thyroid gland, and supra-renal capsules are particularly small; but the contrary has in some instances been noted.

[†] Communication des Cavités Droites et Gauches, p. 340.

(*i*) Intellectual activity and aptitude for acquiring knowledge rank low in cyanotic children: deficiency of this kind has been almost invariably observed, when the child has survived sufficiently long; still this rule is not without its exceptions. The congestion of the brain and cord, arising in the quasi-asphyxial state of the fits of dyspnœa, often issues in convulsions,* somnolence, and even temporary coma. Reflex phenomena, such as jerking action of the limbs and grinding of the teeth, are frequent during sleep.

(*k*) Cyanotic children usually exhibit a sad, suffering expression of countenance; decrepit, and prematurely old-looking, they care little for the ordinary amusements of their age, dreading the occurrence of a paroxysm through the slightest exertion or exposure to chill.

691. *Physical signs.*—The conditions in a case of cyanosis which may conceivably give rise to morbid physical signs, are the cyanæmia itself, the malformation, and the disturbed dynamism of the heart generated by both.

(*a*) Now, does cyanæmia induce murmur? Is it possible that the constitution of dark blood is *per se* a sufficing cause of soniferousness in movement, and that herein lies the hitherto ignored, or at least one of the ignored, causes of "venous murmur" [240]? The answer must be in the negative; for in the most intense form of cyanosis, where the whole system is fed with very little else than black blood, as in cases of the arterial transposition above referred to, there need not be, and is not, of necessity, any murmur at all audible in the cardiac region.

(*b*) Next, as concerns the malformation, the physical signs indicated must, of course, vary with its precise nature; and it is remarkable enough that, even in the gravest varieties (take, again, as an instance, the arterial transposition), the structural elements of murmur may be wholly wanting.

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* J. R. Reynolds, in his elaborate and scientific work (Epilepsy, p. 230), contributes three examples of cyanosis attended with convulsions.

(*c*) Thirdly, few malformations, even aided by the morbid crasis of the circulating blood, maintain persistently a state of dynamic disturbance of the heart. Increased impulse of disturbed rhythm is essentially a paroxysmal occurrence.

(*d*) It seems advisable to warn the student, farther, that none of the malformations producing cyanosis, alter of themselves, the areas of deep or superficial cardiac percussion-dulness. If those areas be increased, sequential hypertrophy or dilatation, or both, have been at work.

(*e*) In the most common variety of malformation connected with cyanosis (namely, an open state of the foramen ovale and constriction of the orifice of the pulmonary artery, with sequential hypertrophy, simple or eccentric, of the right ventricle), systolic basic thrill, doubtless in the pulmonary artery, may be felt [63, *a*]; the systolic murmur of pulmonary constriction may be traced in the supra-basic regions up to, about, and beyond the second left cartilage; and the signs of an hypertrophous state of the right ventricle will be coincidently found. In fact, all the signs of pulmonary stenosis will be distinctly marked [637]. The pulmonary murmur may be exceedingly loud,—I have known it audible at a short distance from the chest-surface.

(*f*) We seem scarcely in a position to assert positively, that patency of the foramen ovale may ever, of itself alone, cause murmur; almost invariably in such records as I have met of the coexistence of the two things, either constriction of the pulmonary orifice is actually stated to have existed, or it may have existed,—all mention of the vessel being omitted by the narrator. On the other hand, no satisfactory evidence exists that patency of the foramen proves invariably, and under all circumstances, incapable of inducing murmur. It is, of course, certain the foramen may be even large enough to admit the point of the little finger, without a particle of murmur occurring;* but, *per contra*, it is very well conceivable that the tension, or other conditions, of the edges of the opening might, in particular cases, lend themselves, with unusual ease, to sonorous vibration. Strength of current, we know by experience, is not, *under otherwise favouring circumstances*, requisite for the production of murmur. And Hughes

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* For example, the case of transposed vessels, Med. Chir. Trans. (vol. xxv.), already referred to; patient ætatis 10 months.

Bennett[*] suggests he may have, and W. O. Markham maintains he actually has, heard a murmur thus generated. In a slightly cyanosed child, aged four years, a rough *systolic* murmur (therefore coincident with the diastole of the auricles), audible about the heart's base and the left infra clavicular region, indistinct below the nipple, scarcely to be caught at the heart's apex, loudly defined at both sides of the upper half of the interscapular space, seemed inexplicable, *post mortem*, except as the result of struggle of the blood at an open foramen ovale. The communication from the right to the left auricle was sufficiently large to admit the point of the little finger; and there was no constriction of the pulmonary artery, nor any other condition of heart, conceivably explanatory of the murmur.[†] The theoretical synchronism of an *intra-auricular* murmur may be made matter of dispute; but, admitting with Tilbury Fox,[‡] that probably the moment would be *pre-systolic*, is it not very likely a murmur really of that rhythm might have been mistaken for one of systolic time?[§] Fox suggests, by inference, that the murmur might have depended on coarctation of the arch of the aorta; but in time it ought then to have been *post-systolic.*

(*g*) Some while since I heard a murmur in a little girl, aged ten, the subject of cyanosis, in what may be called its *intermittent* form, differing in site and mode of conduction from any recognized variety. The murmur was medium-pitched, harsh, of maximum intensity in the sternal infra-basic cardiac region, but faintly conducted either upwards or downwards, and inaudible in either vertebral groove. When I examined the child, there was scarcely any cyanotic tint except at the inner canthi; but under excitement or from bronchial irritation, to which she was subject, the parents stated, the surface became livid generally.

I suspected at the time, and a case published since[‖] gives much surety to the idea, that this murmur might by possibility be *intra-ventricular*, from deficiency of the septum. In J. Johnson's case (as in a similar one recorded by Peacock) the murmur was "most distinct in the horizontal position, and frequently disappeared when the boy was raised upright,"—an excellent illustration of the general proposition, set down in a former place (p. 80), that disappearance of a murmur under the influence of change of posture supplies no proof of its inorganic nature. In J. Johnson's case the pulmonary orifice scarcely admitted a probe; but the murmur, heard during life, was not that distinctive of pulmonary stenosis, and seems really to have been *intra-ventricular*.

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[*] Clinical Medicine.

[†] Brit. Med. Journal, 1857.

[‡] Medical Times and Gaz., Sept., 1859.

[§] This supposition appears to me also justifiable in the instance of another narration of the kind (Mackey, Brit. Med. Journal, Dec., 1871, p. 666). Here, neither the part of the cardiac origin, where the murmur attained its greatest strength, nor its mode of propagation, are mentioned.

[‖] J. Johnson, Brit. Med. Journal, Sept., 1872.

692. *Period of Origin and Duration.*—Cyanosis, symptomatically understood, is far from being always congenital. Of 101 cases of malformation, attended with well-defined symptoms, in 74 these were noticed at or very shortly after birth; in 15 they appeared before the end of the first year, and in the remaining 12 at various periods, from ætatis 1 to 14.* Stillé's return gives 40 congenital, 31 non-congenital, in a total of 71.† There cannot be the smallest doubt, however, that in many cases where the symptoms have first attracted attention at an advanced period of childhood, they had not then come into existence for the first time, but merely undergone striking increase.

It has been suggested, by different classes of theorists, that when cyanosis is non-congenital, and attracts notice for the first time in early childhood, enlargement of the heart gradually opens out the foramen ovale, and so induces the intermixture of venous with arterial blood; or that congenital constriction of the pulmonary artery increases. In the adult, the appearance of cyanosis has sometimes been apparently traced to a blow, a fall, or an effort; possibly some forcible separation of the edges of the foramen ovale had occurred. Sometimes ulcerative openings in the auricular or ventricular septum have seemingly proved the immediate cause. Occasionally the external evidences of the malformation have been but slight, until the accidental occurrence either of endocarditis or of serious bronchial or pulmonary inflammation: the mechanism in these two cases is, of course, different.

Once established, cyanosis, as a rule, to which there can be but few exceptions, remains a permanent condition, though its exact amount may vary from time to time, ranging, in truth, from a tint, so slightly abnormal, that the existence of the disease may not strike the eye on casual observation, to that of the deepest-coloured claret. Such cases might be distinguished as *intermittent*.

Tilbury Fox ably argues, on the evidence of the following case, that cyanosis may, under special circumstances, be *temporary*. A child became very darkly cyanosed on the eleventh day after birth, —no other cyanotic symptom existed, and no abnormal sound was detected at the heart. In forty-eight hours the intensity of the discoloration had yielded; in a week no trace of it remained, its disappearance having been attended with the changing hues of ecchymotic absorption and slight jaundice. Fox tenders the following explanation: the ductus arteriosus and foramen had become on the eleventh day, that of the appearance of the discoloration, less and less patent,—but probably advancing to complete closure with less than usual rapidity. By the closure, first of the duct, then of the foramen, all the blood of the right side of the heart was compelled to travel through the pulmonary artery, which now presented some "obstruction," partly congenital, partly due to the lateness of the normal changes in the duct and foramen; after a little time the "obstruction" was overcome, and the cyanosis removed.* But if the postulated "obstruction" existed, why was there no murmur; why were there neither dyspnœa nor cardiac distress? Jaundice, vomiting, serious hæmaturia, and, above all, extravasation of blood into the skin, shown by the changing hues of the hæmatin, as described, are strange accompaniments of cyanosis. Altogether the case is singularly mysterious.

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* Peacock, *loc. cit* p. 118.

† Amer. Journ. Med. Sc., N. S., vol. viii., 1844.

693. *Relationships.*—Laennec had a notion that cyanæmia in some measure antagonises tubercle: Rokitansky, pushing the idea to extremes, has taught that cyanosis, like other states in which venosity of the blood predominates, "offers a complete protection" against tuberculisation. Numerous cases are on record showing the fallacy of this doctrine absolutely stated;† what degree of antagonism, if any, really exists between the affections, has scarcely been submitted to statistical investigation. As far as such inquiry has gone, it tells wholly against the antagonistic theory; for Peacock finds evidence of active tuberculisation in 16·07 per cent. of cases in which the patient, having malformed heart and cyanosis, survived the age of eight years.‡

Cyanosis seems to have some share of influence in producing pericarditis: but this point also requires further examination [373].

Bizot has shown, in refutation of an old idea, that the foramen ovale is somewhat more frequently closed in phthisical, than non-phthisical persons.*

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* Med. Times and Gaz., August, 1859.

† *E. g.* Louis, *loc. cit.*, p. 313; and Fearnside, Association Journal, March, 1854. Chevers has collected thirteen similar cases (Pulmonary Artery, p. 136).

‡ *Loc. cit.*, p. 137.

694. *Manner of Death.*—Death is usually of slow asthenic type, through imperfect hæmatosis,—facilitated in many instances by pulmonary atelectasis, congenital or acquired, and emphysema. Or cerebro-spinal phenomena of congestive character may abruptly destroy life; serous effusions and visceral hæmorrhages also hasten the fatal issue occasionally. These are almost essential effects of the disturbed condition of the circulation; but, accidentally, death may occur from almost all varieties of acute or chronic disease, in patients who survive the period of early childhood. In some instances the manner of death has not only distinctly been phthisical, but of ordinary phthisical type.

695. *Anatomy and Mechanism.*—The symptomatic state, just described, is known by experience to be connected with various malformations of the heart and perverted modes of origin of its great vessels. The general tendency of the more common of these malformations is to alter the relationships naturally subsisting between the two sides of the heart and the two kinds of blood—dark and florid. But some of the number act in other ways, and the entire series may be referred to three species, as follows:—

A. *Conditions permitting direct communication of the arterial and venous circulations.* (*a*) *In the heárt.*—Open foramen ovale; congenital deficiency of part of the ventricular septum; acquired perforation, of ulcerative or other character, throwing the auricles, or the ventricles, or all the four cavities into, practically speaking, a single cavity; heart formed of one auricle and one ventricle, the latter giving off one artery, which divides into a pulmonary artery and aorta, &c. (*b*) *In the great vessels.*—Freely pervious ductus arteriosus; aorta rising from both ventricles, or from the right ventricle, or from a trunk common to itself and the pulmonary artery; or, as in a specimen now before me,† full-sized aorta rising from a hypertrophous and dilated right ventricle,—pulmonary artery, excessively small and valveless, springing from a rudimentary left ventricle,—ductus arteriosus pervious,—right and left ventricles communicating by a narrow sinus,—connections of pulmonary veins and venæ cavæ with left and right auricle respectively, as in the normal state, &c.

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* Mém. de la Soc. Méd. d'Observation, t. i., p. 360.

† From a patient of Sydney Turner's.

B. *Conditions causing distribution of black blood almost solely to the systemic capillaries, and of red blood to the pulmonary capillaries, without, practically speaking, any intermixture of the two kinds of blood.* Here appear cases where the aorta rises from the right, and the pulmonary artery from the left, ventricle,—the venæ cavæ, as in the natural state, communicating with the right, and the pulmonary veins with the left, auricle;—where, consequently, there are two distinct circulations, communicating alone by the foramen ovale and ductus arteriosus, if (as is the rule), these remain open.

C. *Conditions obstructing the entry of blood into the lungs or intensely congesting them, so as to prevent oxygenation.* (*a*) *In the heart:*—Excessive smallness of the right ventricle; extreme narrowness of the tricuspid orifice: these states disturb the process of distribution of blood *to* the lungs. Great contraction of the cavity of the left ventricle, extreme stenosis of the mitral orifice: these conditions prevent the return of blood *from* the lungs. (*b*) *In the great vessels:*—Partial or complete obstruction of the orifice of the pulmonary artery.

696. Two different theories have commonly been urged in explanation of cyanosis as a dependence on these abnormal conditions:—(1) The venous and arterial intermixture theory;(2) The systemic venous stasis theory.

(1) *Intermixture theory.*—The open condition of the foramen ovale, lending itself to intermixture of the two currents, and, in consequence of frequently co-existing hypertrophy of the right auricle, facilitating flow from the right to the left side, rather than in the converse direction, seemed to afford a ready explanation of the dark tint. This theory of intermixture was long received without question; but against its exclusive adoption the following facts seem to depose more or less emphatically: *a.* Free communication may exist between the blood-currents, as far as the existence of a widely gaping foramen proves this, without cyanosis. *b.* Cyanosis has been wanting, if records may be implicitly trusted, where there was but one ventricle, and where the aorta rose wholly from the pulmonary artery. *c.* When cyanosis coexists with the anatomical conditions of intermixture, no direct ratio holds between the amount of discoloration and the freedom of communication. In a case noted by Louis, where the foramen only equalled a lentil in size, and the orifices and cavities of the heart proved free, more discoloration had existed than in another where the opening was much larger, and the pulmonary orifice

contracted.* *d.* Ribes has related a case where the aorta rose from the right ventricle, and yet cyanotic symptoms did not appear until the age of three years. *e.* The ventricles may communicate and yet the face be pale [883]. *f.* Fouquier observes that the fœtal skin, though always circulating black blood, is not cyanosed.† *g.* In the natural state the colour of the skin comes of the different coloured bloods moving in capillaries and venous radicles lying in close juxtaposition; the effect must be the same as if different bloods were mingled in the same vessels: therefore, as the former condition does not cause cyanosis, there is no reason the latter should do so.

(2) *Venous stasis theory.*—Morgagni started the opinion that cyanosis is really to be ascribed to systemic stasis in the venous radicles, arising from obstruction at the pulmonary orifice. Louis, adopting this view, observes that the blueness of the fore-arm, when ligatured for venesection, does not come of want of arterial blood, for this continues to reach the textures, but from engorgement of their venous system. Stillé, arguing in the same direction, notes more or less marked obstruction of the pulmonary orifice in fifty-three of sixty-two cases. Abnormal septum in the right ventricle, exercising a like obstructive influence, is attended with cyanosis.

But this explanation, also, proves inadequate. For:—*a.* No direct ratio holds between the amount of cutaneous discoloration and the amount of narrowing of the pulmonary orifice. Louis has himself incidentally conceded this, as we have just seen. *b.* This holds true, whether the pulmonary obstruction be acquired or congenital. Nay, more, cases, recorded by H. Roe and others, show there may be no cyanosis at all, from even great congenital contraction of the vessel. *c.* How, too, is this doctrine reconcileable with the fact, that the most intense venous obstruction, intra-cardiac or extra-cardiac, but intra-thoracic, may occur without inducing true cyanotic discoloration? In the course of my experience I remember to have seen but one case of thoracic disease—a very grave example of emphysematous atrophy of the lung—in which the tint of skin was fairly assimilable to that of really deep cyanosis. In truth, the excessive rarity with which an amount or quality of discoloration, at all comparable with that of true cyanosis, occurs as a consequence of organic obstructive disease of the right heart in the adult, has always proved a stumbling-block in the way of those, who support the theory of venous stasis, solely and exclusively, as the cause of congenital cyanosis. Chevers attempts, more ingeniously than convincingly, it seems to me, to get over the difficulty, by suggesting that the special pliant extensibility of the capillaries, appertaining to new-born infancy, is required to ensure the necessary dilatation. But on what ground does he set aside the well-known tendency of the capillaries and venous radicles to augment in calibre with advancing years? He refers to a case in which extreme narrowing of the pulmonary orifice in adult life was unattended with the slightest lividity (*loc. cit.* p. 76).

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* Communication des Cavités, &c., p. 344.

† This argument of Fouquier, concerning the circulation of black blood in the fœtal skin is, however, of very doubtful value. Chevers (*loc. cit.*, p. 79) insists upon the want of facts proving that the blood of the fœtus in utero is less perfectly oxygenated than that in the arteries of the mother.

To resume: It stands, then, an unassailable truth, that the foramen ovale may be widely patent without the occurrence of cyanosis; so, too, the fact is equally well established, that the pulmonary orifice may be congenitally constricted to a high degree, and the skin be of natural colour.* But a satisfactory *tertium quid* may, I think, be moulded out of the two theories. Grant that perforate auricular or ventricular septum and constricted pulmonary orifice coexist, and the occurrence of cyanosis seems to become a certainty †: on the one hand, the labour required at the right side of the heart to overcome the obstacle at the pulmonary orifice, forces of necessity venous blood through the foramen ‡; while, at the same time, the systemic venous stasis, resulting from the pulmonary obstruction, contributes to darken the tint. Probably, too, that tint will prove of the blue or of the claret variety, according as intermixture or stasis severally predominates: the quality of the cutaneous textures themselves, too, will modify the tint; the thinner they are, the deeper the hue. Nor can it for a moment be questioned (on the evidence of existing cases), that accidentally added causes of systemic stasis will greatly deepen the tint.

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* It has been suggested that the absence of cyanosis in such cases of pulmonary stenosis depends on the proportional growth of the right ventricle,—*plus* force of propulsion by a supplementary hypertrophy making up for *minus* quantity propelled. But such hypertrophy exists in cyanotic, as well as in non-cyanotic, examples of the stenosis in question.

† In H. Roe's cases of congenital pulmonary stenosis, unattended by blue discoloration, the foramen ovale was perfect.

‡ If the pressure of the current on either side of the opening be equal, there is no reason why each current should not pass on without commingling at all, or more than very slightly, with the other. Such nicely-balanced pressure doubtless exists in those numerous cases of open foramen, unattended with cyanosis, where there is no constriction of the pulmonary orifice, and no dilated hypertrophy of the right cavities. It is almost self-evident that the admixture must be very free to modify perceptibly the colour of the blood.

The effective power of intermixture, provided the venous quota be sufficiently large, seems clearly proved by cases of transposition of the great vessels, such as that already more than once referred to. In this case the surface was permanently leaden blue in tint, the only arterial blood reaching the systemic capillaries being that carried through the ductus arteriosus. No mechanical obstruction existed in the heart; but the hue grew notably darker under all dynamic influences, impeding the circulation through the lungs. In this instance, too, a slightly corrective condition of the intense cyanæmia arose out of the relative calibre of the great vessels at their origin, as explained in the history of the case (*loc. cit.* p. 12); and it appears to me exceedingly probable, analogous arrangements (as of the bronchial arteries, for instance), indirectly promoting arterialization, may exist in many cases without being detected.

697. *Causes.*—Defective development of the heart is considerably more common in males than females: according to a calculation by Peacock, in the ratio of 57·2 to 42·8. The explanation remains to be found.

Malformation of the kind is sometimes hereditary. All physical defects in her offspring are invariably referred by the mother to influences, mechanical or emotional, occurring to herself during pregnancy; but there is no evidence, approximating even to proof, that congenital vices in the heart are thus produced.

To fœtal endocarditis may commonly be traced obstruction at the pulmonary orifice; certain valvular affections are similarly engendered during intra-uterine life. For the history of errors of development the reader is referred to works on Teratology.

698. *Diagnosis.*—(*a*) In the new-born infant the only affection, with which it appears possible to confound cyanosis, is apoplexia neonatorum (intense congestion with actual extravasation of blood into the membranes of the brain and spinal cord), clinically characterised by great lividity of face, swollen scalp, feeble action of the heart, slow irregular respiration, clenching of the hands, convulsive actions, torpor, chilliness—conditions lapsing into fatal asphyxia, unless treatment, especially bleeding from the umbilical cord, prove successful. But the tint of skin in cyanosis is different,—bluish, not livid; the scalp is not swollen, nor is there general tumidness of the upper part of the body; the action of the

heart is rather in excess than deficient in strength; and the respiration is not laboured, irregular, and slow.

(*b*) In the young adult the distinction is more difficult. It may well happen that a slight amount of malformation, insufficient in itself to produce cyanosis, may have existed at birth, and eventually proved the cause of blue discoloration, either through its own increase or through the contingent aid of some acquired obstructive disease of the organs of circulation or respiration. How is cyanosis of this mechanism to be distinguished from discoloration wholly due to acquired disease? Marked blueness, rather than lividity of tint, will depose in favour of partially congenital origin; but a positive diagnosis will prove unattainable.

(*c*) The specific vice of form, affecting the heart or vessels, can in some varieties be at least strongly surmised, if not actually announced with certainty. Thus, narrowing of the pulmonary orifice with a defective auricular septum (the most common of all cyanotic malformations) may be diagnosed with much security, when a systolic basic thrill, accompanied by murmur, transmitted to the left supra-basic region, and scarcely audible posteriorly [112], coincides with well-defined cyanosis.* As life advances, the physical evidences of right-sided hypertrophy would further strengthen the diagnosis.

(*d*) It would appear, that a systolic murmur of maximum intensity in the infra-basic space, between the third cartilage and the apex of the heart, and coexistent with cyanosis, might be accepted as proof of perforate ventricular septum [691, *g*].

(*e*) If a child were deeply cyanosed at birth, and no murmur could be heard, we should be justified in strongly suspecting that the aorta and pulmonary artery were transposed; or that some other grave vice, throwing no physical obstacle in the way of their blood-currents, affected the great vessels at their origin. (*Vide* also 901.)

699. *Duration of life and prognosis.*—The duration of life varies with the nature of the malformation: but the *degree* rivals in importance the *form* of the vice in the heart. And experience amply shows, that the issue depends also on extrinsic conditions, independent of form and degree of malformation. Hence there is no security of prognosis. An infant with moderately constricted pulmonary artery may perish almost at birth; while, it is certain, life may be prolonged to the twelfth year, though the vessel be absolutely impervious. Again, recorded cases of transposed great

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* See a valuable illustration by S. T. Speer, Med. Times and Gaz., Oct., 1855.

vessels show the malformed child may struggle on to the near completion of the third year, or perish within the first few days of extra-uterine life.

Some practical guidance may be had from the degree of frequency of the suffocative paroxysms.

700. *Treatment.*—The treatment of a case of cyanosis resolves itself into the prevention, as far as possible, of paroxysms of dyspnœa and palpitation. Tranquillity of the circulation, by the avoidance of all emotional excitement, mental or bodily, and of all conditions likely to congest the lungs, the liver, and the abdominal organs, is to be aimed at; the temperature of the skin maintained by warm clothing, moderate exercise, and friction; and that of the body, generally, raised, if the stomach be not disordered thereby, by the free consumption of oil, fat, gum, and other aliments of respiration.

According to the testimony of Gintrac, alcoholic fluids produce drunkenness with unusual rapidity in, and are otherwise very injurious to, cyanotic persons: this is easily intelligible as a result of their imperfect respiration. The fact reminds one of the observing landlord, who noticed he could at will alcoholise his guests with small or large quantities of wine, according as he ventilated their dining-hall ill or well.

Where this is attainable, a warm dry climate should be resorted to.

§ XIV.—INJURIES AND WOUNDS OF THE HEART.

FOREIGN BODIES.

701. Foreign bodies, more particularly needles, sometimes enter the tissues of the heart, either directly through the chest-wall, or from the œsophagus.

PERFORATION BY NEEDLES.

702. The progress of events may be of various kinds, when a needle enters through the chest into the cardiac region.

(*a*) In a remarkable case, where the needle had perforated the pericardium, death followed in about fifty-eight hours, through hæmorrhage into the pericardial sac, from laceration of about half an inch square of the apex of the left ventricle by the point of the foreign body.*

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* H. Wright, Brit. and For. Med. Journal, Nov. 13, 1869.

(*b*) But the course of events may be much slower. Thus, in a circumstantially recorded case, the foreign body first entered the right mamma; a month later, while the patient was stooping to reach something from the floor, it penetrated more deeply, causing pleurisy; four months later she had pneumonia and bronchitis of the right lung; a month afterwards "spasms of the diaphragm," which were succeeded by obstinate vomiting and subsequently by pain about the heart and pericarditis. The needle was found after death, reaching from the right ventricle into the left.*

(*c*) Again, it would appear a needle, at least five centimètres long, may be fixed in the ventricular septum, protrude into each ventricle, and be coated with fibrinous concretion at both ends, without giving rise to any symptom. It is even affirmed no murmur could be heard, in the case specially referred to—a statement the more remarkable, as there was fibrinous exudation on the pericardial surface.†

(*d*) A man for nine days followed his ordinary occupation in pain and discomfort, having a needle fixed in the apex of the heart. On the ninth day G. W. Callender made an incision over the fifth intercostal space, seized the exposed eye of the needle and withdrew it. The man recovered perfectly.‡

BULLET AND SHOT WOUNDS.

703. Twelve cases have been collected by S. S. Purple § in which the patient lived fourteen days with a ball in the pericardium. "Dr. Hopkins, of Ohio, has reported a case in which the patient survived fifteen days with a pistol-ball encysted in the wall of the left ventricle. In a case recorded by Dr. Randall, of Ohio, the patient died on the sixty-seventh day, and three shots were found in the right ventricle and in the right auricle, the wounds having cicatrised. In the 'Indian Annals of Medical Sciences' a man is said to have survived ten weeks with a musket ball in the cavity of the left ventricle. Fournié records the history of a man, who, wounded by a ball, fell as if he were dead. Three months afterwards, he suffered from severe palpitations, which nearly disappeared after three years. He died six years after the receipt of the injury, from some unconnected malady;

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* Leaming, Brit. and For. Med. Rev., July, 1845.
† Piorry, Union Méd. 1858.
‡ Lancet, Feb. 22, 1873.
§ New York Medical Monthly, May, 1855.

and the ball was found lodged in the right ventricle, near the tip, and resting on the septum."

Yet more extraordinary was the heart of a patient aged forty-four, containing a bullet at its apex, which had been lodged there from a musket-wound received when he was fourteen years old. Six weeks after the injury he returned to work. Later on he married. His last illness was ascribed to cold. The ball was surrounded by atheromatous deposit, the heart dilated, but not hypertrophous.*

It is important, medical jurists should know, that the heart may be ruptured by a pistol-shot without any, but the slightest, injury to the skin. In a rapidly fatal instance of the kind, the skin was not broken, no rib was fractured, the pericardium remained entire, and yet the intercostal muscles and left ventricle were perforated.†

WOUNDS BY CUTTING INSTRUMENTS.

704. Though this subject be essentially surgical, a few observations may be ventured on concerning the medical bearings of injuries of this class.

705. *Symptoms.*—The symptoms vary with the character and direction of the wound, though not to so great an extent as might be anticipated: pallor, faintness, or complete syncope; uneasiness or actual pain about the site of the wound, increased by respiration, and rather a dependence on the injury to the external soft parts than to the heart's own structure [259]; external hæmorrhage, very slight,—or more serious, if an intercostal artery be wounded, or if a large external opening coincide with effusion of blood into the pericardium, effected prior to clotting in the actual wound of the heart itself; general anxiety, clammy perspiration, and other symptoms of collapse. The collapse is commonly due in the main to nervous shock, in part to actual loss of blood. The pulse may remain regular, though notably more frequent than natural; a normal pulse-respiration ratio of 4 : 1 may be maintained.‡

706. *Physical signs.*—If effusion of blood occur into the pericardial sac to any extent, its presence will be revealed by the sudden supervention of the physical signs of fluid accumulation,—

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* Hamilton, Brit. Med. Journal, Jan. 19, 1867.
† Ward, Med. Times, January, 1862.
‡ Boswell Reid, Lancet, Dec., 1855.

with tendency especially to the peculiar triangular-shape of percussion-dulness already fully described [p. 44]. Friction-murmur will, at first, be inaudible, though a peculiar rumbling noise may be detected. If life hold on, the signs of pericarditis may follow.

707. *Diagnosis.*—But though the diagnosis may in the majority of instances be established on the evidence now briefly reviewed, taken in conjunction with the character of the external wound, the depth to which the cutting instrument may have penetrated, &c., still occasionally the utmost difficulty may be felt in determining whether the heart's fibre have really suffered or not. On the one hand cases are recorded, where many of the symptoms, habitually indicating actual wound of the organ, existed, yet it had wholly escaped; and on the other hand, as a single example of the negation of direct evidence, a case recorded by Dupuytren may be referred to. In this instance a man survived the infliction of several penetrating wounds of the right ventricle for upwards of three weeks, the pericardial sac meanwhile containing a large quantity of blood; and yet neither general collapse, nor any single local symptom, pointing to injury of the heart, occurred. But significant physical signs must, it would seem, have existed under the circumstances.

707*. *Prognosis.*—Wounds of the heart may be instantaneously fatal, when on a large scale; they may be rapidly fatal through hæmopericardium; or they may be more slowly fatal through secondary inflammations and intra-cardiac thrombosis, followed by distant embolism.

Or temporary respite may occur; and after a few days' quietude sudden death ensue, either from detachment of a clot or probably from direct injury to the still fresh wound, through incautious exertion or even emotional excitement of the organ.

Yet, further, there are a few cases on record proving the possibility of complete recovery, after even very serious wounds of the heart. A man was discharged from hospital "cured" on the seventy-eighth day after receiving a stab from a knife two inches below the left nipple, close to the sternum: the symptoms and signs clearly proved implication of the heart's substance. Dying, *nearly twenty years later*, with symptoms of hypertrophy and valvular disease, evident cicatrices were found at the antero-inferior part of the right ventricle, in the septum of the ventricles, and in the mitral valve.* Systolic thrill and powerful murmur, detected during the acute period, were referred to the opening in the ventricular septum [691, *g.*]

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* Brugnoli (of Bologna), Presse Méd. Belge, July 20, 1862.

708. *Treatment.*—In the treatment the careful avoidance of too free stimulation on the one hand, or too lavish use of antiphlogistic measures on the other, seems to be the chief matter for consideration. On the whole less danger is to be apprehended from primary collapse, than from consecutive fever and some forms of cardiac inflammation.* Rest is all-important, and should be constantly maintained for days and weeks, after all apparent local effects have passed away. From inattention to this rule, sudden fatal syncope has been known to occur upwards of three weeks after the accident.

Cardiac sedatives, especially aconite, should be employed, if there be any notable excitement of the circulation.

Fibrinous or muscular inflammation must be met by the cautious use of antiphlogistic remedies. Mercurials are recommended; but seem deserving of no confidence.

If extravasated blood stagnate, especially with added serous effusion, within the pericardium, paracentesis of the sac seems a reasonable measure; the results of the operation in scorbutic pericarditis, indeed, justify a hopeful view of its probable success.

If evidences of intracardiac thrombosis, or of distant embolism, occur, the treatment presents great difficulty. Solvents of thrombal masses, if effective, would probably disintegrate likewise any coagulum in the wound.

§ XV.—STRAIN OF THE HEART.

709. Strain of the heart, produced by various kinds of effort, acting on the organ slowly, feebly and enduringly, or quickly, powerfully and temporarily, may produce various kinds of mischief, statical and dynamic, ranging between actual rupture of its walls and slight disturbance of motor and sensory innervation.

710. Though it be questionable, whether actual muscular rupture is ever thus produced in a heart thoroughly sound texturally [672]; it is, on the other hand, certain that all the other less disastrous effects of overstrain, to be by-and-by enumerated, including, in great probability, even tearing of a valve or tendinous cord, may be thus brought about in hearts free from structural disease,—and this, not only as far as evidence goes, but as far as reasonable surmise suggests.

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* Purple, New York Med. Jour., May, 1855, p. 420.

711. Cases of overstrain of the heart are clinically of acute and sudden, or of chronic and gradual development.

712. (I.) *Acute form.*—I shall only refer here to cases, where the effect, at the time of the extra strain on the heart's texture, falls short of causing organic mischief.

713. *Symptoms.*—The chief symptoms are various anomalous sensations in and about the cardiac region,—a feeling as if something had given way; faintness to actual syncope; general muscular tremor; perspiration.

The action of the heart, slackened at first, may almost immediately become frequent, though regular, and (as I have actually known) beat 120 per minute persistently for months,—without, it may be mentioned, the occurrence of exophthalmos. Or the rhythmic movement may be specially affected, and the pulse become excessively irregular.

A sufferer from these symptoms is unfit for the ordinary occupations of life, and the temper becomes irritable and captious, or downright morose.

714. *Duration.*—Dynamic disturbance of this kind may hold on for years without the intervention of appreciable organic change.

715. (II.) *Chronic form.*—Cases of slow development may be divided into two sub-classes: those in which symptoms of some kind or other begin to appear from the actual time of the overstrain; and those in which their occurrence is deferred for a variable length of time after the cause has presumedly begun to act.

716. In cases of slow development the affection may be purely dynamic, or, as is more common, be attended with organic change.

717. The organic changes thus induced are: overgrowth, dilatation; perverted nourishment, inducing cirrhotic and fatty changes; and valvular disease.

At least valvular disease seems to claim a place here, because patients occasionally present themselves with defect of a valve, in whom the blemish is known not to be congenital, and who have never had rheumatism, gout, syphilis (either hereditary or acquired), or idiopathic cardiac inflammation, but who had, years before, gone through muscular exertions felt at the time (though not precisely in the heart) to have over-taxed their powers.

718. *Causes.*—Mountain-climbing, pedestrian feats, undergoing the training process for rowing matches, throwing great weights, excessive gymnastic exercise, *especially the use of the club*, and intemperate coition are the forms of abuse of strength, which

have most frequently fallen under my notice as causes of overstrain of the heart.

719. *Treatment.*—The knowledge of the cause, when possible, is of course essential: the effects are to be managed, according to their nature, on the principles laid down in other parts of this work.

§ XVI. CARDIO-THYROID EXOPHTHALMOS.

720. Among the numerous people, who seek relief for dynamic palpitation of the heart, a certain small share are found to suffer in addition from enlargement of the thyroid gland, excited action of the cervical arteries, and staring protrusion of the eyeballs. This strange combination of conditions—so strange that their association would seem a mere accident—forms, on the contrary, an established entity, a *quatuor juncta in uno*, holding a well-defined place in clinical experience. Known by a variety of names, the complaint will be described under the title suggested above, as fixing on the mind the three most essential of its four component elements.

721. The clinical history of cardio-thyroid exophthalmos has been worked out by various observers with great precision, since the first inkling of its existence was given by Flajani in 1802 [*]; but the completion of its natural history awaits the establishment of the link that binds the four phenomena together. For, in truth, no one of the series of necessity causes, or of necessity is caused by, any other: equally true is this of any binary union of the four elements. There must then exist some factor beyond all four, either acting as their original and essential cause, or as a necessary condition of the activity of the real cause. But of this more by-and-by.

722. In the most perfect cases, clinically considered, all four units of the tetrad are associated. But in less complete examples there are either: (1) palpitation and cervical throbbing; (2) palpitation, cervical throbbing, and thyroid enlargement; (3) palpitation, cervical throbbing, and protrusion of the eyeballs, without enlargement of the thyroid [†]; or (4) overgrowth of the thyroid, followed after a certain time by prominent eyeballs, and eventually by palpitation and cervical throbbings [‡]. The bronchocele must

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[*] Collezione d'Osservazioni, 1802, t. iii., p. 270.

[†] The occasional absence of bronchocele was long since noted by Prael, Archiv f. Ophthalmol. 1857.

[‡] *E. g.*, Morell Mackenzie, Trans. Clin. Soc., vol. i., p. 13, 1868. [Vide 729.]

either in the latter class of cases (the rarest of all) be of a specific character, or the two conditions must be merely coincident: else how comes it that in ordinary goître no such sequential changes arise?

723. *Symptoms.*—Assuming that two or more units of the tetrad exist, as the basis of the disease, the condition of the various functions will be found as follows:—

(*a*) The frame is generally thin, sometimes actually emaciated, where the disease has lasted long; the sleep bad, in some cases excessively so; the countenance, more or less disfigured according to the amount of displacement of the eyes, commonly wears a dejected, morose expression; the temper habitually grows irritable, and the spirits depressed.

(*b*) The skin, occasionally of anæmic tint, may, as I have twice seen it, be cyanæmic; but in these instances both sides of the heart were dilated, and tricuspid reflux existed to a slight extent. Bronzing of the skin, analogous to that of "Addison's disease," has in rare instances been observed*; a maculated eruption of the scalp was noticed by Trousseau.

There is no true pyrexia; but a generally diffused subjective sense of heat (an important item in the symptoms, as regards the theory of the disease) is often complained of: this may be connected with free or clammy perspiration,—a condition assimilable to the "vapours" of women ceasing to menstruate. The objective temperature, as first shown by Cheadle,† ranges somewhat above the par of health,—reaching in one of his cases 101° in the axilla: the rise of temperature is found to be bilateral, and also equal in the face and axillæ. There is no clubbing of the finger-ends.

(*c*) Nothing special is noticeable in the condition of the joints, bones, or muscles of the limbs.

(*d*) Inordinate appetite is of pretty frequent occurrence; there is no dysphagia; primary digestion goes on without hindrance; diarrhœa has been so often noticed, that the idea of connection with the disease suggests itself.

The liver gives no particular sign; the spleen may often be shown by percussion to be enlarged.

(*e*) Subjectively a sense of fulness in the chest is the only noteworthy thoracic symptom; the breathing is not necessarily hurried, and I have not seen mechanical interference with the trachea (as is common in ordinary bronchocele), prove a source of distress,—the swelling is in truth rarely large. Paroxysmal dyspnœa and a feeble, cracked condition of the voice may occur from pressure on the recurrent nerve. Epistaxis has occasionally been noted.

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* Begbie, p. 10, Transl. of Virchow, "Struma Exophthalmica."

† St. George's Hospital Reports, vol. iv., p. 183, 1869.

(*f*) The heart-palpitation is habitually of the excited, frequent, abrupt, violent type, distressing, but unattended with acute pain [317]. The beats habitually number over 100 per minute, and may reach 140—160; but the rhythm may continue perfectly regular, though sometimes, as originally noted by Flajani, irregular.

The heart itself, natural in bulk at first, in rare instances becomes dilated and hypertrophous, as time wears on. Valvular mischief, if present, is purely accidental: I have not met with pericarditis [374]. The heart-sounds are, as a rule, normal.

The inferior thyroid arteries, the carotids, the vessels of the enlarged gland, and sometimes the temporals, pulsate more or less violently, with objective thrill. In the gland itself the pulsation is attended with a diastolic swell. All this is subjectively very wearying to the sufferer. Flushing of the face (bilateral, as far as I have seen), is of frequent occurrence.

The auscultation-results may be singular enough. Over the general mass of the enlarged thyroid intermittent, medium-pitched blowing murmur may be heard, accompanied by a loud, lower-toned, continuous hum; while in the jugular district, at the side of the gland, whistling and roaring hum almost drowns a powerful carotid whiff. The disease has, in the instance where these sounds are audible, lasted several years.

These patients are occasionally distressed by dynamic pulsation of the abdominal aorta [753]. Generally speaking, the vessels of the limbs are free from enlargement or excessive pulsation; almost all observers insist on the relative smallness and quietude of beat of the radials. But J. W. Begbie is nevertheless right in affirming, that the vascular system throughout occasionally pulsates to excess.*

The pulse-respiration ratio undergoes perversion,—a ratio of 120 : 20 or 6 : 1 proving not uncommon.

The pulse-tracing will vary with the amount of anæmia and the force of the heart's action at the moment; nothing distinctive can be expected in the sphygmogram.

The superficial veins over the enlarged gland are distended.

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* Vascular Bronchocele, &c., p. 14, 1863.

The jugular veins full, but non-pulsatile (unless independently of the disease) are occasionally the seat of distinct hum.

The blood is certainly sometimes spanæmic; but in one of the best-defined cases I have chanced to meet with (a female aged about twenty-five), floridness of hue and deficiency of venous murmur proved the absence of anæmia. The state of the blood, in fact, calls for further investigation.

(*g*) I have seen the lymphatic glands under the jaw (and once in the axilla) notably large; but no established relationship holds between glandular engorgement and the complaint.

(*h*) The evidence, as far as collected, shows the urine is unaffected; but frequent micturition is sometimes complained of.

(*i*) Neither is there any necessary defect or disturbance in the genital functions. The catamenia may be perfectly regular; still irregularity has not only been observed, but has been distinctly connected with variation in the entirety of the cardio-vascular symptoms.

The mammæ of a male sufferer from the disease were found in one case greatly enlarged,—the left organ hard, congested and painful, yielded colostrum.*

(*k*) The scalp is sometimes tender to the touch, and throbbing cephalagia, sense of fulness in the head, and vertigo may exist. The temporal arteries may be over-sized and forcibly pulsatile.

Nothing noteworthy occurs, as a rule, in regard of motor function. The "weakness of the lower extremities, amounting almost to paraplegia, observed in two or three aggravated cases" by J. W. Begbie, seems wholly exceptional. In a remarkable case by Morell Mackenzie (*loc. cit.*) epileptiform convulsions and mania occurred; but it is more than doubtful whether the brain-disease, on which these depended, can be looked upon in any degree as a final evolution of the nerve-disorder probably essential to cardio-thyroid exophthalmos.

These patients are generally dull intellectually. They form an exaggerated estimate of their disfigurement and of the danger of their disease, which probably leads to the perversions in their moral feelings and affections not very unfrequently observed.

(*l*) Hysterical symptoms have occasionally been noticed; but the disease may exist without the slightest evidence of the kind.

(*m*) Tinnitus aurium, with a sense of disagreeable throbbing in the ears, and slight deafness, annoy some patients.

The eyeballs prominent and staring, to an amount varying with the force of cardiac action, subjectively feel full, but are not the seat of pain. The protrusion, as a rule bilateral and equal in amount on the two sides, may be unequal; or, as observed by Prael, unilateral (on the right side), in three of nine cases.

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* Von Basedow, quoted by Virchow, *loc. cit.*, p. 9.

The amount of protrusion may be sufficient to show the conjunctiva upwards of quarter of an inch round the cornea, and expose the insertion of the recti. Generally of slow production, the prominence may occur suddenly, as from a violent fit of coughing and retching.* Bodily fatigue may temporarily greatly increase the protrusion.

There is no nystagmus, and no squinting; but diminished power of co-ordinating the movements of the eyelids, when the eyes are directed downwards, has been detected by Von Gräfe and M. Mackenzie.

The eyes look moist; tears fall over the cheeks. Singularly enough, the open state of the eye, though maintained even in sleep, entails, as a rule, neither inflammation nor even injection of the conjunctiva,—the inevitable results in the somewhat analogous case of unclosed lids in paralysis of the portio dura. Still I have seen conjunctivitis; ulcerative keratitis also does sometimes occur, and the eye has been damaged in some instances by sloughing inflammation.

Gentle and continued pressure will push the eyes back into the orbits; but when the pressure is taken off, they slip forwards again.

The sight is habitually unaffected, and the power of visual accommodation maintained. Still some patients complain of enfeebled vision, "mist before the eyes," and muscæ volitantes.

The pupils have been found in very various states: natural, statically and dynamically (this is most common); sluggish; enlarged; contracted. No unilateral peculiarity has been recorded.

The ophthalmoscope habitually fails to show any defect in the optic disc. The choroid is sometimes injected.

(*n*) Facial neuralgia has been noticed (Stokes, *loc. cit.*); but so seldom, that any real connection between the two states seems unlikely. A sort of unilateral brow-ague has fallen under my notice.

(*o*) The thyroid gland, seldom of very large dimensions, though strikingly above par in size, nodulated, elastic, soft at first, but disposed gradually to harden, is the seat of throbbing expansile thrill, general or limited, varying in amount at different times, and gradually lessening, as the enlargement diminishes in favourable cases tending towards cure.

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* Adams, quoted by Stokes, Dis. of Heart, p. 295, 1854. The protrusion came on in a single night, without any fresh exciting cause having occurred, in a case recorded by R. Taylor, Med. Times, May, 1856, p. 516.

The acoustic conditions within the thyroid area have already been described [723, *f*].

724. *Course.*—Habitually the affection runs an irregular course,—exacerbations being connected with disordered uterine function. Improvement of all the conditions of the disease may occur to such an extent as to simulate complete recovery,—relapse ensuing with or without apparent cause. Temporary increase of exophthalmos, and even passing increase of bulk of the thyroid gland, sometimes occur in the early stages from excitement of the heart. Pregnancy and child-bearing often control, and sometimes even promote the removal of, the disease.

On the other hand, the affection may advance steadily to a certain point, and then stand still,—or it may pursue a uniform course either to recovery or to death.

Commonly commencing slowly and insidiously, the outbreak may be sudden, and the early progress acute. The exophthalmos especially may form, as we have seen, in a few hours. I have known initiatory palpitation follow immediately on a miscarriage.

725. *Duration.*—There is little to be said of a positive kind as to the possible duration of the disease. In one case which fell under my notice, palpitation and exophthalmos had existed certainly for ten, possibly for more, years.

726. *Mode of death.*—Cardio-thyroid exophthalmos does not destroy life *per se*,—more or less grave structural alteration in the heart commonly contributing to the fatal issue. The observations of Stokes lead to the conclusion, that death ensues "from anasarca and pulmonary congestion as in dilated heart." But cases are recorded showing that death may be the result of the pure disease, either through slow failure in nutrition and diarrhœa, or more rapidly through cerebral disturbance.

In the latter category appears the case recorded by M. Mackenzie (*loc. cit.*, p. 15), in which Lockhart Clark found the corpora quadrigemina and medulla oblongata much softened, and a drachm or two of sanguineous fluid in each lateral ventricle.

727. *Anatomical appearances.*—It can scarcely be said the affection possesses any anatomical character in the true sense of the term; but dissection has revealed the following conditions

holding a more or less determinate relationship to the actual complaint.

Thyroid gland.—The enlargement of the gland seems essentially to depend on hypertrophy of its structure, with enlargement of the veins and arteries, which are thickened and tortuous. The size of the mass (as we have seen), is never very great.

In some cases, however, cystic development occurs, with intervening fibro-plastic strata, as in ordinary goître.

Thymus.—This organ has sometimes been found still present in the adult sufferer.*

Spleen.—Although the spleen has pretty frequently been enlarged, weighing in one instance twenty ounces,† it is doubtful whether the fact has any real significance.

Eyes.—Opinions explanatory of the condition of the eyeballs may be placed in two groups: in the one the eye is supposed to be changed in structure; in the other, simply driven forwards, or allowed to slip, from its socket.

In the first group we find simple enlargement of the eyeball, increase in quantity of the aqueous and vitreous humours, hydrophthalmos, severally suggested.

In the second the list is longer, and consists of: enlargement of arteries; distention of veins, with coagulated blood over the eyeball (Keith); loss of tonicity of muscles of the eyeball (Dalrymple); fatty metamorphosis of these muscles (Von Recklinghausen); some essential change, occasionally hypertrophy, but mostly hyperæmic swelling, of the orbital fat (Virchow); pressure on the veins of the cervical muscles (M. Hall); congestion of the arteries of the eye, but still more that of the ophthalmic veins (J. W. Begbie).

As far as evidence appears to me to go, the increase of orbital fat is accidental, while the protrusion essentially arises from arterio-venous distention. Probably too, as argued by Aran, the influence exerted by the sympathetic on the orbital muscle of H. Müller, the action of which is to carry the eyeball forwards, may play a part in the extrusion of the globe.‡ The notion of its dependence on hydrophthalmos, or other organic change in the eye itself, has long been relinquished.

Heart.—The heart is either perfectly free from textural disease, or the seat of various changes in nowise connected with the essence of the complaint: the former condition is the rule. The prolonged overaction much less frequently entails hypertrophy, than might be expected.

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* Markham, Trans. Path. Soc., 1858.

† W. Begbie, *loc. cit.*, p. 17.

‡ *Ibid.*, p. 19.

Arteries.—The inferior thyroid is greatly enlarged, while the superior retains its natural diameter. The aorta is not oftener atheromatous than in the mass of people dying at the same age. The temporals undergo enlargement.

Nervous system.—The lowest or the middle and lower cervical ganglia have in some cases been found enlarged, hard and firm on both sides. Keith affirms he has seen them tuberculized. Again, the trunk and ganglia of the sympathetic have been found simply and purely atrophous; or, along with the recurrent nerve, injected and infiltrated with serum. The medulla oblongata, we have seen, may be diseased: but whether the two things were connected clinically, or not, is doubtful.

728. *Causes.* (*a*) *Predisposing.*—The affection is certainly not endemic; in this respect differing from simple bronchocele, soil and climate have nothing to do with it. It scarcely occurs before, but often at, the period of puberty, and during and at the close of gestation. Greatly more common in females, it has so rarely been seen in the male, that Flajani's earliest recorded case has had few fellows. One of the most remarkable of these will be found in Cheadle's paper. It occurs both in single and married people, and is often connected with uterine derangement. I must note, however, that in, markedly, the worst case I ever saw (the patient was single), the uterine functions were perfectly healthy. Graver, in the abiding intensity of its cardiac element especially, when anæmia (as it often does), co-exists, there may not be the slightest evidence of any such defect in the blood [723, *f*]. The complaint occasionally afflicts two members of the same family—a fact not without significance, in view of its absolute rarity. Dark and fair people seem pretty equally affected; but, as the proportions of the two tints in the populations furnishing the cases is unknown, the fact remains barren of inference.

(*b*) *Exciting.*—The observed exciting causes have been fright; long-sustained emotional excitement; over-work, mental and bodily; continued effort (thus, I have known a grave form of the disease produced by mountain-climbing). Hysterical seizures, be they ever so severe and continued, have not appeared to me capable of generating the complaint.

729. *Nature.*—The nature of the disease can scarcely be considered established. If, on the one hand, it be true, as it actually

is, that palpitation usually forms the first link in the chain, on the other, that palpitation may hold on as an abiding torment for years without sequential prominence of the eyeballs, is a fact familiar to all. So, too, extreme enlargement of the thyroid may exist without association of palpitating heart or prominent eyeballs. The *primum mobile* of the malady manifestly lies deeper: there is a something beyond its surface-phenomena. Now the morbid anatomy of the affection plainly suggests, what physiology also would teach, that disturbed influence of the sympathetic and the cervical ganglia on the vaso-motor system must play an important part in its completed pathogenesis. But then comes the difficulty, as well shown by Cheadle, that the clinical grouping of the symptoms does not accord accurately with the physiology either of paralysis or hyper-excited action of the cervical sympathetic. This difficulty will, however, probably be removed by more intimate acquaintance with the functions of this portion of the sympathetic, as well as of the disease clinically considered; so too, of the further stumbling-block, presented by the bilaterality of the phenomena at the clinical outset of the disease. But whether there is, yet beyond this, a diseased state of a certain portion of the spinal cord (the cilio-spinal tract), connected with the ganglionic nervous system, the heart and the cervical vessels, as forcibly maintained by Laycock, is undetermined. The view held by J. W. Begbie, that the action of impoverished blood on the vaso-motor nerves of the sympathetic is the real root of the disease, seems irreconcileable with the clinical facts, that no trace of anæmia is in some cases discoverable,—while, on the other hand, anæmia may exist for years, may even destroy life, without affecting in the least the bulk of the thyroid or the position of the eye-balls.

In attempting to fix the pathogenesis of this affection, there is yet another fact that must not be lost sight of, namely, the occasional occurrence of morbid prominence of the eyeballs, independently of any other element of the ordinary tetrad. A few cases have come before me in the course of years, in which, in association with uterine disturbance and *very slight* anæmia, the eyes have become as distressingly prominent as in any case of the complaint, we have been considering, though the thyroid gland was of normal size and the heart and cervical arteries models of healthy action.

730. *Diagnosis.*—It seems impossible this peculiar affection can be confounded with any other; the day has long passed when, as in a case referred to by Stokes, it could be mistaken for aneurism.

731. *Prognosis.*—The complaint gets well in a small proportion of cases, especially where not carried to extremes. The prognosis is most favourable, when the patient is distinctly anæmic, and where emotional disturbance, physical strain, catamenial disorder and pregnancy seem to have played a part in its production.

732. *Treatment.*—The treatment consists in removing, as far as possible, any apparent cause of the disease, and indirectly tranquillizing the circulation. Under various obvious circumstances, iron is the most important remedy, and, in conjunction with veratrum viride, has more than once in my hands freed the patient from any real distress,—the thyroid gland at the same time undergoing decrease in size and increase in hardness.

The local use of iodine and of Kreuznach-extract (so valuable in common bronchocele) has appeared to me to exercise no influence on the condition of the thyroid, still less on the other phenomena of the disease. But Cheadle reports a remarkable case (*loc. cit.*) in which a state of great comfort, if not of actual cure, was effected by a nine months' course of iodine internally and externally. Palpitation here distinctly preceded the enlargement of the thyroid. In other instances, referred to by him, iodine either proved slightly useful, or did no harm.

The constant application of iced water to the surface is reported to have sometimes done good service,—and in the acuter stages counter-irritation appears to have lessened the bulk of the gland.

The local use of electricity has rarely been followed by these notable changes, not unoften observed in the treatment of common goître. M. Meyer, however, reports four cases where "brilliant results were obtained by galvanizing the cervical sympathetic for a considerable time." *

The proposal to name this disease after some observer who had strikingly contributed to the elucidation of its history, seems difficult of adoption: Flajani, Parry, Von Basedow, Graves, Stokes, Begbie, would all on different grounds be entitled to the distinction.

§ XVII.—THROMBOSIS, EMBOLISM, AND INTRA-CARDIAC THROMBI.

733. Spontaneous coagulation of the blood within its reservoirs and channels, and translation of particles of coagula from their original site to some more or less distant spot in the circulating

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* Medical Record, p. 10, January, 1873.

system, where they become impacted, play a large part in the Natural History of disease, and may, in their general bearings, be conveniently considered in the present place.

734. The story of the manner in which existing knowledge on the subject of spontaneous coagulation and its possible issues has been acquired, is not a little curious. It adds one more to the numerous examples of the resuscitation, in the guise of novelties, of old ideas. The facts are referrible to two orders,—those bearing on the coagulation-process, and those particularly referring to the distant wedging of detached fragments of clot.

Vague notions on both phenomena may be traced from a very early period. Even Galen seems at the least to have strongly suspected the grave part occasionally played by solidifying blood within the vessels. Nearly two centuries ago Gould described, with singular minuteness, the effects of coagulation within the sinuses of the dura mater, and even sought to affiliate some of the symptoms of rheumatism to capillary impaction.* Again, the idea of the translation of particles of coagulum from one spot in the circulating system to another, and its fixture within the latter, is put with unmistakeable significance by Van Swieten. A passage, from this author's lucid review of the causes and mechanism of apoplexy, thus completely and succinctly embodies the whole doctrine: "Quæcumque *sanguinem*, lympham, materiem spirituum ita mutant, *ut per arterias cerebri libere ire non possint, sed impacta hæreant;* talia sunt sæpe,—*Polyposæ* in carotidibus, vertebralibusque arteriis *concretiones, sive circa cor primò*, sive intra ipsum cranium, *factæ.*" †

Nevertheless, if these views ever made any impression, it certainly was short lived. All ideas of the kind faded out of memory for half a century,—when the new, or modern, period of this history seems to have been initiated by Legroux. In 1827 Legroux published two cases of gangrene of the hand and fore-arm, presumedly caused through instantaneous obliteration of the brachial artery by a fragment detached from a coagulum in the heart; he further generalised the idea of the migration of particles of clot through the entire arterial system.‡ He was followed by Allibert in the following year with the account of a case of gangrene of an upper extremity, caused by arterial occlusion with particles identical in character with coagula found in the left auricle.[*] Next appear the observations of Louis (1837), of Lediberder (1837), and of Baron, whose essay, incorporating cases by all three, was printed in 1838.[†] Then follows the evidence given by John Taylor in his Lectures on Pericarditis, showing that exudation-products, borne from the right ventricle into the pulmonary artery, might effectually plug its minute branches, as well as the capillaries of various viscera.[‡] After this comes the contribution of Paget, the earliest general account in our language of the coagulation-process in the pulmonary artery.[§]

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* Philosophical Transactions, p. 546. 1684.

† G. van Swieten, Comment. in H. Boerhaave Aphoris. (Apoplexia), t. iii., p. 258. Ed. Parisiis, 1771.

‡ Thèses de Paris, 13 Août, 1827.

But it must be confessed, that, despite these various contributions, and the significance claimed for them by their authors, the general doctrine, they so plainly taught, made but little advance. And it was reserved for Virchow in 1846-7, while applying the old terms *thrombosis* and *thrombus* to the coagulation-process, and resulting coagulum, and inventing those of *embolus* and *embolism* to signify the detached fragment and the circumstances of its detachment and circulation, to finally establish the place of these phenomena in pathology.[‖]

735. *Mechanism of thrombosis.*—The more important conditions, under which arrest of the blood-flow may result in the formation of a thrombus, may be tabulated in the following manner :—

BLOOD-STASIS AND THROMBOSIS.

SITE.	MECHANISM.	
Intra-Cardiac.	Obstruction in pulmonary capillaries,—chiefly pneumonic.	
	Stagnation from failing cardiac expulsive power, tending to *asystolism*,—caused, for example, by prolonged nausea (p. 402).	
	Inflammation, endocardial ;—*directly* from roughening of surface ; *indirectly* from embolic obstruction of pulmonary capillaries caused by itself.	
	Certain conditions of the blood,—especially hyperinosis.	
Intra-Arterial.	Inflammation, with roughness and exudation-products.	
	Fissures ; cracks ; unevennesses ; calcareous concretions, lamellar, spicular, &c.	
	Mechanical Influences.	Intrinsic—Tumour-pressure.
		Extrinsic—Ligatures, needles, mechanical pressure, &c.

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[*] Recherches sur une occlusion peu connue des vaisseaux artériels, &c. Thèses de Paris, Avril, 1828.

[†] Archives Gén. de Médecine, Mai, 1838.

[‡] Lancet, 1845,—orally earlier [408, *i*].

[§] Med. Chir. Trans., 1844.

[‖] In seinem Archiv. Later came the observations of Kirkes, on the lodgment in the cerebral vessels of fibrinous particles borne through the circulation from the left side of the heart (Med. Chir. Trans., 1852).

- Intra-Venous.
  - Obstructed circulation.
  - Altered composition of the blood : in anæmia ; phthisis ; convalescence from acute diseases, as pneumonia ; the puerperal state.
  - Failing cardiac power, combined with other causes of sluggish circulation—as in pulmonary artery [854].
- Intra-Capillary.
  - Diathectic blood-states—Rheumatism, purpura, scurvy.
  - Morbid poisons, *e.g.*, glanders, pyohæmia, gangræmia, septicæmia, &c.
  - Following on embolic impaction, caused by
    - Fragments of distant thrombus.
    - Particles of exudation-matter or hard vegetation.

736. The effects of thrombosis vary in all conceivable degrees. Thrombosis may be instantaneously fatal (or at least associated with instantaneous death), when its seat is the pulmonary artery ; prove rapidly fatal, when occurring in the cerebral veins and sinuses ; it may produce a simulation of paraplegia, when arising in the abdominal aorta ; cause sudden, extreme, and intensely painful swelling of the lower, or more rarely an upper, limb, in the course of phthisis, or during convalescence from typhoid (Pyerian) fever, followed by more or less enduring hardness and over-bulk of the part ; or, from the peculiarity of its locality and the slightness of its amount, its effects, if any, may be practically untraceable.

737. Thrombi forming within the heart and in the pulmonary artery are the only varieties of blood-clot, that require special notice in this volume : to the description of the former we at once proceed ; an account of the latter will be found elsewhere [853].

INTRA-CARDIAC THROMBI.

738. Coagulation of blood within the heart may occur either after death, or *in actu mortis*, or during life, and at various periods more or less distant from its actual close. The characters of the resulting coagula are for the most part well-defined, and clearly significant of the date of their formation, whether *post mortem*, *intra mortem*, or *ante mortem*. But it must be confessed that instances, on the whole exceptional, every now and then occur, wherein it appears impossible to fix with certainty the period, at which the coagulation-process really set in.

739 (A). *Post-mortem variety.*—To this variety belong masses of variable size, consisting commonly of a stratum of loose, gelatinous and more or less watery-looking,* pale straw-coloured, semi-transparent fibrine, coating (commonly on the upper surface),

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* Separation of serum from the clot very certainly continues after somatic death,—the degree to which it occurs probably varying with numerous conditions at present unknown.

or not, grumous currant jelly-like coagulum of variable consistence,—the two being easily separable by washing, when both are present. Structureless, non-laminated, interlaced or not among the musculi pectinati and columnæ carneæ, but non-adherent to the endocardial surface, free from even the earliest indications of an attempt at vascularisation, these concretions are notably more common on the right than the left side of the heart.

Or, if at the period of death the blood have been notably deficient in fibrine, as in all hypinotic diseases; or, if its fibrine have been of non-plastic quality, as in various heteræmic acute specific diseases, of extremely adynamic type; or in alkaline poisoning, as long since shown by Magendie;* or, if the manner of death have been calculated to obstruct coagulation, dark grumous currant-jelly looking masses may alone be found,—distinct separation of the fibrine not occurring at all.

And, further, the clotting process may fail to take place even in an inceptive or imperfect manner: the blood may remain wholly fluid, as in cases of death by lightning; by suddenly fatal nervous shock caused, for instance, by a violent blow at the epigastrium; or by chloroform. Suddenness of death seems one element in entailing this condition of the blood,—but obviously only one.

(B). *Variety formed shortly before and in the act of death.*—Fibrinous coagula formed shortly before death are non-watery, firm, opaque, either laminated, and fibrillated, or granular or lumpy, non-uniform in tint, being in some parts pale fleshy-looking, in others yellowish, or slightly greenish; marked on the surface with blood-spots or even quasi-vascular looking streaks, more or less firmly adherent to the endocardium, and becoming rough and shreddy in the process of separation therefrom. Occasionally they completely fill the compartment, they occupy. They may be of uniform consistence throughout their mass: or if the diathetic conditions of the individual, the manner of death, or the atmospheric state be favourable to rapid physical and chemical change, the fibrine may soften, even become fluid, centrally; and under the latter circumstances bear the most deceptive naked-eye resemblance to pus.†

To these characters Richardson has added the presence of a simple or spiral groove on the outer surface, or a central canalicula, caused by the blood-current.*

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* Lectures on the Blood, Lancet, 1839.

† Vide Art. Adventitious Products, Cyc. of Anat. and Physiology, p. 114, 1848; here will be found the distinctive marks between genuine pus and softened fibrine. It seems of late years to be forgotten that the distinction in question is as old as the day of Dupuytren.

Now, precisely in proportion to the degree of perfection of these characters will the certainty arise, that the coagula presenting them, were really formed before death; and the conviction will be strengthened, if they are found on both sides of the heart. But on the other hand, I am persuaded coagula having all the characters of the first variety (A), are sometimes produced immediately prior to and during the final struggle.

And there is a very striking appearance in some of these coagula which, though habitually an index of *ante mortem* formation, may, I think, be met with in rare cases of *post mortem* coagulation. I mean that perfect cast of the pulmonary valves which is occasionally seen on the exterior of a thrombus filling, closely grasped and surface-moulded by, the infundibulum of the right ventricle, the pulmonary artery and both its main divisions.† I demur to the commonly received opinion, that such moulding necessarily signifies formation prior to death, because I have found it distinctly defined, where the manner of dissolution had been irreconcilable with the notion of cardiac thrombosis.

(C). *Variety formed at a more or less distant period before death.* —The appearances of this variety vary considerably. Sometimes the concretion consists of very firm stratified fibrine, more or less markedly vascularised, and so firmly adherent that it can only be removed by the scalpel,—the endocardium invariably undergoing injury in the process.

Or, again, the concretion occurs in the form of masses of various shapes, globular, elongated, ovoid, sessile, or pedunculated; ranging in size from a pin's head to a walnut and upwards; in colour whitish, pale dirty straw, greenish, yellow red, or reddish yellow; in structure obscurely fibrous, laminated, granular, and lumpy or amorphous; invested with a membranous coating identical in physical attributes and continuous with the adjacent endocardium. This variety of concretion is not vascularised; it has been supposed to undergo nutrition-changes through imbibition from the surrounding blood. Saline precipitation, and even a rude imitation of bone-formation, may occur interstitially within its substance.

The variety just described is solid, and of more or less closely uniform consistence throughout. But there is another form in which, while the globular outline and mode of endocardial attachment are identical, the concretion is hollow and sac-like. The contents of the sac are fluid, like water and blood mixed, or lees of wine, or of yellowish-green hue and puriform look. Sometimes the interior cavity is sub-divided, giving a multilocular aspect. The cause of the sacculated appearance is simply the softening of the central portion of the original solid fibrinous mass,—the colouring matter of the blood, associated in different amounts, giving the various tints to the fluid. The wall is often very thin, and its actual rupture, with effusion of the contained fluid, has in rare instances been observed. These saccular concretions may occur in any of the heart's cavities; they are said to form most frequently in the left ventricle,—it has occurred to me to observe them oftenest in the auricles, attached to the appendices. They are, absolutely speaking, rare in all situations. They may be attached to the general tract of the endocardium or to its valvular portions. In the latter case, frequently pedunculated, their varying relationship to the connected orifice may give rise to very peculiar physical signs [616].

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* On Fibrinous Depositions in the Heart. 1860.

† Univ. Col. Museum, No. 3636.

(D). Lastly, fibrine may also be deposited from the blood at various, more or less lengthened, periods before death, in the form of small wart-like masses, or so-called *vegetations*. The most frequent seat of these bodies is on the surface and at the edges of the aortic and mitral valves; but, though exceedingly rare at the right side of the heart, there is no part of the endocardial surface on which they are not occasionally seen. Smooth or jagged, rounded or amorphous, they adhere either to an apparently unbroken endocardial surface, or to fissures in the membrane, or amid the columnæ carneæ and the cordæ tendineæ.

Once deposited from the blood, the fibrine undergoes inspissation and hardening in the majority of instances,—though the clinical history of cases sometimes seems to prove, they may maintain an unchanged condition of softness for years. It does so in this way: a given murmur has existed for years,—the patient dies of an independent affection, and to explain the murmur a soft fibrinous vegetation is found on the surface of a valve. In some cases they, on the contrary, become firmly fibroid or pseudo-cartilaginous in aspect, or undergo calcification or pseudo-ossification.

740. *Causes and mechanism.*—Whatever be the ultimate form assumed by a cardiac concretion, it originates simply in coagulation of the blood. In some cases the process is sudden and

effected on a large scale,—in others slow and of very limited extent.

The conditions promoting the necessary coagulation are referrible to: (*a*) obstructed movement of the blood through the heart; (*b*) anomalous composition of the blood; (*c*) endocarditis.

(*a*) Obstruction of the intra-cardiac current may result from all varieties of valvular impediment; from general dilatation and all conditions tending towards *asystolism;* from local pouching and aneurism; from mere roughness of surface, caused by fissuring or otherwise; and from pressure, especially on the right side of the heart, by mediastinal tumours or sacculations of the aorta. Although from the feebleness of contractile power of a fattily metamorphosed heart, stagnation, with sequential clotting of the blood, might be expected to be of frequent occurrence, it actually proves, so far as I have seen, to be rare: a fact probably explicable by some peculiarity in the crasis of the blood. In exhausting diseases of various kinds, provided the fibrine be not seriously reduced in plastic quality, coagulation may take place towards the close of life.

(*b*) Among the anomalous conditions of the blood favouring the clotting-process, foremost stands the hyperinosis of sthenic inflammations and certain specific affections. In rheumatic fever, croup, and especially in pneumonia (as originally insisted on by Bouillaud) the chances of serious obstruction in the right heart, through the formation of non-soluble thrombi, add materially to the other sources of danger common to those affections. Separation of the fibrine is also favoured within the heart in certain forms of poisoning of the blood, as in common pyohæmia, or in that sequential to glanders. In anæmia, just as spontaneous coagulation occurs in the veins of the lower extremities and in the sinuses of the dura mater, so is it occasionally, though rarely, effected within the heart.

(*c*) However observers may vary in their estimates of the frequency with which noteworthy clotting occurs in acute endocarditis, the mechanism of its occurrence is plain enough. Roughness of surface, produced by deposition of lymph, and occasionally by minute fissuring; the hyperinotic crasis of the blood; the poisoned condition of that fluid arising from the inflammation-products, which are thrown into the general current from the endocardial surface; and the obstruction of circulation from valvular difficulty, all contribute their share in effecting the result.

741. *Effects.*—Cardiac thrombi produce certain effects, when fixed to the surface; they produce others through their separation in totality or in fragments therefrom.

(1.) *Attached but partially mobile.*—The effects of thrombi, when in this condition, essentially obstructive, vary with their size, their precise situation, especially in respect of the orifices, and their rapidity or slowness of formation. Nothing can be more singular than the degree to which large masses may be tolerated within the heart, provided their growth has been a work of time. The case of huge encephaloid tumour of the right auricle, referred to in a former place [588], where no single cardiac symptom had ever occurred, until, from altered relationship of the mass to the orifices of the auricle, fatal obstruction was instantaneously produced, sufficiently illustrates the statement. The projection of an intra-auricular recent thrombus into the tricuspid orifice, in such manner that it becomes impacted, may cause instantaneously fatal syncope. I have known this occur without any real separation of the obstructing portion of the clot from its main mass.

The singular manner in which a state of orifice, permitting reflux, may be converted by coagulum-deposit into one of pure obstruction, is elsewhere proved [636].

If a coagulum be, as it were, semi-fixed *in situ* by a long pedicle, it may float in and out of an orifice, in such fashion as now to obstruct the onward current, now to cause reflux [616].

(2.) *Detachment and embolism.*—If a coagulum of some size be *separated* from its attachments, constant risk of its becoming impacted in an orifice, so as to cause sudden and fatal obstruction, ensues. The fact has been observed at the mitral orifice.* Such detachment may take place, while the coagulum is yet moderately soft, or after it has undergone more or less complete calcification.

More frequent is the detachment of minute fragments of soft clot or firm vegetation, which, borne into the blood-current, either pulmonary or aortic, eventually reach a spot, beyond which, from the narrowness of the vessel, their further progress becomes an impossibility. Under these circumstances one of two things happens: either, after having impeded the local circulation for a time, the minute coagulum breaks up, dissolves, passes on and leaves the interior of the vessel free; or the little fragment remains fixed as a permanent plug, exciting inflammation, coagulation, or hæmorrhage, according to the special textural conditions of the spot. Such are the general conditions of *embolism* springing from the heart.

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* Van der Byl, Lancet, January, 1837.

742. *Physical Signs.*—The physical signs of abrupt and extensive intra-cardiac thrombosis are, on the whole, sufficiently characteristic, especially when taken in association with their conditions of origin. It is to be remembered, certain of these signs are principally to be found on the right side of the heart, as the ordinary seat of the clotting process.

(*a*) The impulse may retain the characters existing prior to thrombosis; if regular, it may remain so: or, as is more common, sudden irregularity occurs, affecting force as well as rhythm.

(*b*) Extension of percussion-dulness, mainly to the right of the sternum (I have known it reach beyond the nipple), is invariably demonstrable, where the heart's previous outline had been well ascertained.

(*c*) Obscurity of the normal sounds—a confused state of their toning—marked inequality in their strength with successive beats of the heart, signify the difficulty of the blood-movement through the organ. These peculiarities are better defined on the right than the left side.

If murmur have pre-existed, this may be intensified, lessened, or annulled: all will depend on the precise relationships of the clots, the orifices, valves, and tendinous cords: or new murmur, or murmurs, obstructive or reflux, or both, may form. Of such *de novo* murmurs, the systolic, basic, and infra-basic variety, transmitted to the left supra-basic region, and indicating obstruction both in the infundibulum of the right ventricle and at the pulmonary orifice, is by far the most common.[*] Greatly less frequent (I have not, for my own part, met with a positive example), is the parallel murmur indicative of obstruction in the left ventricle.

(*d*) Acute emphysema of the lungs is, according to the observations of W. B. Richardson, clinically demonstrable as an effect of the deficiency of blood in the capillaries of the alveoli. He seems to admit, however, that signs of acute emphysema are almost limited to childhood,—being exchanged for those of congestion in the adult (op. cit. p. 16).

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[*] I greatly regret to find myself at variance in my statements regarding thrombal *de novo* murmur, with so earnest and skilled an observer as W. B. Richardson (op. cit., p. 16). But, as his negation of its occurrence seems to be based, not on clinical observation, but on the idea that "the conditions are not such as to produce murmur," I cannot help stating that I have heard new murmur, where *post mortem* examination proved the absence of any other condition capable of generating it, and therefore believe in it. On the other hand, I willingly concede, that murmur is much less frequently met with, than a superficial consideration of the circumstances might lead us to expect.

743. *Symptoms.*—The symptoms of intra-cardiac thrombosis must of necessity vary with the rapidity with which the process is effected,—with the localization and bulk of the resulting thrombi,—and with the local and systemic state of the individual at the time of its occurrence.

(*a*) The symptoms of clot-formation, when occurring in its gravest form, as an evolution of endocarditis, have already been set forth [414]. We need not here detail them again: suffice it to add that the quality of the symptoms will be essentially the same, no matter under what circumstances coagulation takes place acutely on an extensive scale.

(*b*) In less urgent cases, both as regards the actual amount and rapidity of coagulation and the condition of the heart prior to its occurrence, the symptoms are much less grave. An increase in the amount of pre-existing chest-symptoms, especially of dyspnœa, dusky lividity of surface, distension of the external jugular veins, and increased frequency, with variously perverted rhythm, of the pulse, representing the tumultuous action of the heart, ensue. Spongy tumidness of the base of the neck, such as that habitually significant of pressure on the intra-thoracic veins by tumour or aneurism, is sometimes observed; or actual œdema may take place in that situation,—or, on the contrary, be first seen about the feet. Whatever amount of general distress may have pre-existed undergoes more or less marked increase.

Continual nausea and vomiting have occasionally been observed for several days before death, in persons whose hearts contained coagŭla evidently formed during life: they have been looked on as symptoms of coagulation. But the mode of connection of the two things is not clear; concretions may certainly form without disturbing the stomach, while unquestionably prolonged nausea will promote intra-cardiac stagnation and clotting of the blood.

744. *Duration of life.*—Plainly it must be impossible to lay down any general rule concerning the time life may hold on, after thrombosis has commenced within the heart. Everything will depend on the conditions of the process.

(*a*) If coagula form so as to obstruct the right infundibulum and orifice of the pulmonary artery, death is known, as matter of occasional fact, and whether as effect or coincidence, to have occurred instantaneously. But what brief time the thrombus, found in the vessel, may have taken to reach the bulk, efficient for syncopal, and perfected pulmonary, obstruction, is unknown: no guide is to be obtained from symptoms.

(*b*) Even where a thrombus is not *per se* fatal at once through its manner of localization, cessation of life, in cases clearly fatal through the influence of rapid and extensive clotting within the heart, generally follows very shortly on the process. I have known death occur in sixteen hours after the physical signs first distinctly indicated new intra-cardiac obstruction, proved *post mortem* to have depended on coagula. Life is rarely prolonged beyond the third day. But we must remember, that the *onus* of destroying life is in these cases commonly shared by some grave pre-existing disease, endocarditis, pneumonia, &c. The actual consummation of death may with these antecedents, also, be eventually instantaneous,—from syncope caused by effort or even slight movement.

(*c*) Where, on the other hand, the process is slow and on a limited scale, life may be prolonged considerably,—nay, if the formation, even in certain situations of most perilous influence, be very slow, existence may not be demonstrably shortened by the occurrence.

(*d*) Very large solid formations, as we have already seen, form within either auricle, and either instantaneously destroy life through change of position, without having given any previous indication of their presence; or induce anomalously associated pulmonary, cardiac and systemic symptoms for months preceding the fatal issue, itself eventually caused by a fit of mixed asphyxia and syncope.*

745. *Diagnosis.*—The local signs and general symptoms of nervous shock to the heart, the local signs and systemic effects of intra-cardiac obstruction, *plus* the existence of the blood-states already enumerated [735], make up the groundwork of diagnosis in cases of extensive and serious coagulation.

It is almost unnecessary to say that in the characters of the murmur or murmurs, arising out of the obstruction of thrombi, there is nothing specially distinctive. The circumstances, under which the murmur occurs, furnish the main guide to its mechanism. Its systolic rhythm and manner of transmission, either in the line of the pulmonary artery or of the aorta, are obviously valueless as exclusive evidence.

Thrombosis on the left, much less frequent than on the right side, differs symptomatically from the latter in its tendency rather to congest the pulmonary than the systemic capillaries. The difference is, so far, precisely the transcript of what is observed severally in cases of chronic mitral and tricuspid impediment. But going further than even tricuspid reflux, thrombosis of the right heart may eventually almost wholly deprive the pulmonary capillaries of blood.

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* Vide case by H. Douglas, Ed. Med. Journal, April, 1868.

In cases of acute endocarditis, I know of no means by which coagulation can with surety be distinguished from rupture of a valve or ulcerative destruction or rupture of a tendinous cord. The effects of cardiac shock are the same in all three cases; so are the physical signs identical: indeed this must be so, as clotting to greater or less amount will of necessity occur around the injured spot of the valvular apparatus.

For the mode of determining the time of origin of endocardial murmurs I must refer to a former place [417].

746. *Prognosis.*—The prognosis of coagulation within the heart is, as far as existing knowledge guides us, grave in the extreme. In truth, it has not occurred to me to witness more than two or three cases, where the diagnosis appeared to be established with a fair amount of surety, and where disappearance of the symptoms and physical signs, with present recovery of the patient, took place. And even in these instances, inasmuch as the diagnosis could not be esteemed wholly unopen to objection, my evidence of recovery is not so satisfactory as might be desired.

747. *Treatment.*—The measures to be adopted in cases of endocarditic thrombosis have already been described [419, *b*]. And these measures may be had recourse to in all acute inflammatory diseases, where coagulation occurs, especially on the right side of the heart. Where the left cavities are the seat of stasis, I should expect more advantage from free dry cupping of the chest generally, than from any other local agency. In chronic diseases with languid circulation, the risk of coagulation should be averted as far as possible by abstaining, at least in any quantity, from the use of nauseant medicines and digitalis, the obvious effect of which must be to slacken the circulation. Blood-letting should also be refrained from, unless under some urgent necessity. The influence of diluents, recommended for the purpose of rendering the blood less coagulable, can scarcely be trusted to,—and by producing a watery plethora they may do more harm than good.

748. Small quantities of carbonate of ammonia and of bicarbonate of potass, taken two or three times a day, seem to me the

most promising prophylactic,—perhaps the chief practical inference, secured to therapeutics through Magendie's tremendous destruction of canine life, in illustration of his Lectures on the Blood.

But the crasis of the blood must of course always be taken into consideration; the iron and fibrinous diet essential in spanæmia would be utterly out of place, where a disposition to hyperinosis could be traced.

DISEASES OF THE GREAT VESSELS.

SECTION I.—AORTA.

§ I.—SIMPLE DYNAMIC PULSATION.

749. Aortic pulsation, or abdominal or epigastric pulsation or palpitation, as it has been variously called, is a peculiar functional affection of the aorta, essentially constituted by more or less throbbing action of the vessel. Although very positively observable in the thoracic aorta (I have met with it in the arch of the aorta, the innominate and right subclavian), the affection is best known in the abdominal division of the vessel.

750. *Symptoms.*—(*a*) Pulsation at the epigastrium, more or less constant, but aggravated by various influences, such as brisk movement, nervous excitement, irritation of the bowels, or constipation and dyspeptic disturbance, is, as its title indicates, the prominent feature of the complaint. In rare instances, complete intermission of the throbbing action may occur from time to time,—a fact important in respect of diagnosis. When strongest, the pulsatile movement is attended with a feeling of sickness or faintness, or pseudo-globus; there is no actual pain experienced, except the adjacent parts be accidentally extra-sensitive. The epigastrium, however, always bears pressure worse than in healthy people, and may be extremely tender.

(*b*) The constitutional effects of aortic pulsation are sometimes sufficiently serious: it increases already existing "nervousness;" excites apprehension, or obstinate conviction, on the part of the patient, of the existence of deep-seated organic disease; deprives him sometimes of the power of taking exercise, and interferes with digestion, whence arise loss of appetite and emaciation: and some patients, constantly dwelling on the symptom, magnifying its importance, and, fancying that bystanders must notice it as well as themselves, acquire an utter distaste for all society.

There is no pyrexia; the radial pulse presents no distinctive character; and if there be anything special in the pulsation of the femorals, the peculiarity is equally shared by both vessels.

751. *Physical signs.*—(*a*) In well-marked cases, pulsation may be easily seen at the epigastrium, especially in thin people; rarely at the umbilicus. (*b*) The hand, laid on the surface in the course of the vessel, receives a forcible forward impulse, slapping rather than heaving, jerking, quick, abrupt, without distinctly expansile character, but bounding and free,—varying in degree from an action so trifling as to be scarcely perceptible, to one sufficiently powerful to shake the bed. As a rule, there is no lateral expansion to be felt; but, if hardened tissue lie on the confines of the artery, such expansion may at the least be closely simulated. Unless the blood be anæmic, no thrill is to be caught. The vessel, if the patient be very thin, may be reached on either side by the fingers, and slightly moved laterally. (*c*) The transverse limits of the vessel under percussion are natural; but it must be confessed this is a fact very difficult of establishment, though it may be tolerably easy to assure oneself, there is no *great* extent of saccular or fusiform enlargement: the abdominal wall should first be steadily depressed for a minute or so, all gas and fæcal matters, as far as possible, pushed sidewards, and the outline of the artery then carefully sought for by percussion varying in firmness and direction of stroke. (*d*) Aortic pulsation may exist in the highest degree without murmur,—a single systolic* impulsive sound being alone audible; or a systolic and a diastolic sound may be heard, the latter the fainter greatly of the two. Or, what is more common, a single systolic blowing murmur, prolonged slightly, rough and sharp, whiffing or whipping in quality, is perceived,—the more marked, the greater the pressure exercised on the front of the vessel. Such murmur may, however, in rare instances, be heard in the lumbar region behind. Until lately, I supposed that the discovery either of double or of diastolic murmur, in the course of the aorta, positively indicated disease in its coats; but I now know, by a case observed with great interest during life, that there may be intense aortic pulsation, with diastolic murmur, although the vessel be perfectly free from disease, and in calibre below the average. In the instance referred to, however, the pancreas was found, after death, enlarged and hardened, and had pressed somewhat on the vessel in the site of the murmur.†

752. *Anatomical conditions.*—It has been argued by some pathologists, on the ground of the limitation of the abnormal pulsation

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* In applying the terms systolic and diastolic to mark the rhythm of sounds and murmurs in the arteries, I intend to signify synchronism with the systole and diastole of *the heart*, unless the contrary be stated expressly.

† Gosling, U. C. H., Females, vol. v., p. 130.

to certain spots of the vessel, that local organic change must exist. As to positive evidence, there is but little collected. In some of the few *post-mortem* accounts recorded, affirmation is absolutely made that the vessel was in all respects sound; in others, that its walls were flaccid (but as cause or effect of the pulsation?) in yet others, thin. I know from observation it may be texturally sound,—and even one negative case is invaluable in a question of the kind.* Pressure from adjacent parts, if sufficient to reduce the calibre of the vessel, seems very distinctly to promote its undue action. The pulsation may certainly change in precise site from day to day almost; and in rhythm the local throb differs distinctly, sometimes, from that of the heart and of the general tract of the vessel itself. All this points to purely dynamic essence; and the affection seems to consist of an excited state of local motor-innervation, commonly of reflex mechanism. It would be unjustifiable to deny the probability that a morbid state of the vascular coats may occasionally act as the first link in the chain of that reflex mechanism; I have seen well-marked atheroma in a case of the species.† But, on the other hand, there is no necessary connection between such local arterial disease and morbid pulsation; every day's experience exhibits grave calcification of the abdominal aorta, in persons who have been perfectly free from the dynamic complaint.

In some instances the entire of the abdominal tract, and the iliac and femoral arteries are involved.‡

753. *Causes.*—The predisposing and exciting causes of aortic palpitation are sufficiently various. While the complaint is not so common in ordinary hysteria and spinal irritation as might be expected, it accompanies many utero-ovarian diseases, with pelvic and abdominal neuralgiæ; may follow on anæmia of all modes of origin; occasionally attends plethora, sthenic and asthenic; and is induced in some susceptible frames, by green tea, strong coffee, tobacco, and similar agents. Though by no means constant in that affection, it is not unoften observed in cardio-thyroid exopthalmos [723, *f*]. Various disordered states of the chylopoietic viscera produce it—simple dyspepsia, flatulence, and hepatic disturbances among the number; but special proneness to the complaint probably exists, where causes so slight suffice for its generation. Acute inflammation of the stomach, bowels, and

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* Gosling, U. C. H., *loc. cit.*

† Campbell, U. C. H., Females, vol. xv., p. 251.

‡ Davidson, U. C. H., Males, vol. xiii., p. 67.

peritonæum, as insisted on by Stokes, and chronic gastritis, as chiefly illustrated by Faussett, sometimes produce very marked sympathetic action of the abdominal aorta. Pressure of all kinds directly on the vessel takes an important place among its causes; I have known an accumulation of fæces in the transverse colon induce it in an aggravated form.

As far as my experience goes, the affection is quite as common, and as distressing, in males as in females.

754. *Duration.*—Aortic palpitation, once fairly developed, is with difficulty got rid of. Baillie was probably right in the opinion that, though it might vary in amount, it seldom disappeared altogether. The chances of complete cure are greatest, where anæmia lies at the root of the evil; but even here there is no certainty of such result.[*]

755. *Diagnosis.*—(*a*) A very little care will distinguish pulsation of the aorta in the epigastrium from the epigastric pulsation of a downward-displaced heart, or an enlarged right ventricle: it is unnecessary to dwell on the distinctive marks.

(*b*) The real difficulty consists in ascertaining with positiveness whether a given condition of abdominal pulsation be purely dynamic or dependent on a dilated state of the vessel.

Now, first, if genuine, *well developed*, sacculated aneurism exist, the pulsation is expansile, heavy, powerful, slow, and appears kept back, as it were, by some restraint behind it, instead of bounding freely forwards; thrill is occasionally to be felt, and a tumour, with dulness under percussion nearly commensurate with its size, is discovered. A harsh, grating, hollow murmur, systolic, of the characters elsewhere described, audible in front, may also sometimes be detected along the spine posteriorly. All this is accompanied with pain more or less wearing or agonising; and the determination of the true nature of the case, proves, as a rule, sufficiently easy.

But, secondly, to distinguish between *incipient* fusiform dilatation or sacculation on the one hand, and dynamic pulsation on the other is, I believe, often matter of impossibility. A man, aged about thirty, was sent to University College Hospital by Siordet, for an opinion on the nature of the epigastric pulsation under which he suffered. So nearly balanced was the evidence, that I did not venture to pronounce an opinion in one direction or the other. Now the inclination of that evidence, such as it was, told rather for dynamic than structural disease; yet in about eighteen months later the patient came under my notice within a week or so of his death from one of the largest aneurismal sacs I have seen. *Per contra*, there is a case in the hospital books,* in which after very careful and repeated examination, the diagnosis of incipient aneurism of the vessel was set down in dubitative fashion, with a note of interrogation; and though the symptoms greatly improved under the rest and medical appliances of the hospital, they never did so in a sufficiently positive manner to induce me to modify the diagnosis into one of simple pulsation. Now this woman was killed by a street-accident two years later (in August, 1861); and her aorta, though somewhat thin and atheromatous, proved to be wholly free from dilatation.† I find it stated in my notes that, at the close of the patient's stay under my care, the source of impulse was confined to a narrow surface at or near the median line.

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[*] Brackenbury, U. C. H., Males, vol. v., p. 38, March, 1850—a case of this class. I saw this patient again in the autumn of 1853, he was then practically free from the complaint; but he came a third time under my notice, March 15, 1859, and had then been again suffering steadily from it for upwards of four years.

And there are occasional extra sources of perplexity. Thus, where an aorta, healthy in itself, is pushed forwards by an enlarged vertebra, or a tumour connected with the spine—or where an indurated mass lies in front of, or to either, or both, sides of the vessel, lateral expansion may be simulated, the murmur may be harsh, though not grating,—while local pain and general emaciation may be produced by the organic morbid state, whatever it is, superadded to the pulsation.‡ Under these circumstances, the difficulty of diagnosis may be extreme. It appears from the case just referred to, that even the discovery of diastolic murmur in the vessel, that is, synchronous with its own systole, will not prove the existence of aneurism positively; still such murmur very rarely attends inorganic pulsation. As a rule, too, the murmur of the latter origin is inaudible in the loins; but that of aneurism may be similarly limited to the front. The sex of the patient may give accidental aid; for aneurism of the abdominal aorta is very rare in the female, aortic pulsation common,—the gravest examples of functional palpitation I have met with, have, however, been in males. Again, the existence of hæmic murmur in the heart, thoracic aorta, and veins may assist also; but it may tend to deceive too, for the subject of aneurism is not exempted from becoming anæmic.

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* Campbell, U. C. H., Females, vol. xv., p. 251, Sept. 1859.

† I am indebted for the opportunity of examining the aorta to the kindness of Mr. Miller, house-surgeon at the time.

‡ Lucas, U. C. H., Females, vol. xv., p. 312; here simple pulsation was transmitted some inches to the right of the median line through tumour.

To sum up then: in ordinary cases the diagnosis is easy; in some rare instances it will be well to watch the signs for a time before risking an opinion; and to positively affirm the absence or presence of slight peripheral dilatation of the coats of the vessel (fig. 1, p. 476), may every now and then, even after prolonged observation of the patient, remain impossible.

A very remarkable example of aortic pulsation, combined with moveableness of the left kidney once fell under my notice. The most interesting facts of the case were, that when the kidney lay, or was brought by the hand, in front of the throbbing vessel, the proper substance of the organ itself not only seemed to pulsate strongly, but with an action most distinctly expansile to the feel. I learned, in truth, in a manner never to be forgotten, the danger of trusting to the seemingly "expansile" character of any given pulsating action, as distinctive of true aneurismal beat. The peculiarities of each of the two affections in this case threw light on the diagnosis of both; and there was no difficulty in forming an opinion of their nature.

Little, if any, diagnostic help of a sure kind can be expected to be furnished by the radial sphygmograms (*vide* APPENDIX).

Abidingly lowered temperature of the inferior extremities would argue strongly in favour of aneurism rather than of dynamic pulsation.

756. *Prognosis.*—It is inferrible, from the foregoing account, that our promises must be cautious as to the duration or eventual curability of the complaint. But, though it may be sufficiently tormenting to embitter existence, no instance is probably adducible of its having destroyed, or even of its having ostensibly shortened, life.

757. *Treatment.*—In the majority of cases, the treatment of aortic pulsation directs itself to the removal of its causes,—such as spanæmia, spinal irritation, gastric or intestinal disturbance, or leucorrhœa.

The management of the affection in view of its nerve-mechanism should be conducted as follows:—all suspected articles of food, strong tea, and coffee, and similar stimulants should be forbidden, and a plain, nutritious diet rigidly adopted, with but little vegetable or other substances that promote flatulence. An occasional warm aperient, with some carminative adjunct is advisable. Antispasmodic medicines afford relief to the symptom, though they fail to reach its cause; of these, valerian, æther, ammonia, assafœtida, and musk may be employed. Sedatives, such as lettuce, conium, and hyoscyamus, may be given in alternation with others of the class acting more especially on the heart, namely aconite, veratrum viride, and hydrocyanic acid. Digitalis and chloral prove, both of them, though on different principles, servicable. Bromide of potassium, or of ammonium, sometimes exercises a singularly controlling influence over the pulsation, but only for a time. These medicines are beneficially combined with tonics, where the general indications for such agents present themselves.

The application of some four or five leeches to the epigastrium sometimes distinctly tranquillises the pulsating vessel,—even where no fair suspicion exists of the presence of gastritis in any form. Dry-cupping I have sometimes found beneficial, however difficult of explanation the fact may appear. Anodyne embrocations, belladonna plaster, and the endermic use of morphia in the in the scrobiculus cordis, moderate the arterial action.

Change of air and travel, moderate exercise, daily friction of the skin, the shower-bath, sea-bathing, or the tepid salt-water bath, and in fact, all hygienic influences, that strengthen the nervous system and improve the health generally, are among the most effectual agents in the management of obstinate cases.

§ II.—HYPERÆSTHESIA OF THE AORTA.

758. Hyperæsthesia of the aorta may exist in all degrees between downright neuralgia and mere unnatural consciousness of pulsation.

759. There can be no doubt that calcification of the vessel is sometimes the source of severe pain in its tunics, especially when the circulation is excited. Such pain has occasionally been confounded with that of genuine angina pectoris. It has occurred to me, as highly probable, that the peculiar pains felt in the limbs by certain phthisical patients, and which very decidedly do not follow the course of the nerves connected with the spinal system, may depend on excess and perversion of vaso-sensory innervation. They are sometimes spoken of by the sufferers as intermittently or remittently rhythmical.

760. Unnatural consciousness of the action of the vessel (and undue sensitiveness of the kind may extend to a great portion of the arterial system), is a source of annoyance to some hysterical

women. I have known the same symptom, precisely, complained of in anomalous states of the nervous system in the male.

§ III.—INFLAMMATION OF THE AORTA.

ACUTE AORTITIS.

761.—Acute aortitis is a rare disease, at least as far as demonstration of its existence goes; it appears to be singularly uncommon, where it might be frequently expected to occur, namely, in connection with acute endocarditis.

762. *Physical signs.*—The signs of this affection are obscure,—at least in the present state of knowledge. Violent pulsation of the vessel, and tumultuous action of the heart, sometimes exist; it is very probable that, if lymph be deposited to any extent on the lining membrane, thrill may be perceived, where the vessel nears the surface of the chest. In a well marked case, recorded by Parkes,* an extremely loud, rough systolic murmur continued audible from the third dorsal vertebra quite down to the lumbar region—a murmur obviously due to the passage of the blood in the vessel over a surface roughened by patches of lymph. In this instance the pulse was irregular and small, but the aortic orifice was contracted, and otherwise diseased, and the heart in a state of dilated hypertrophy; it does not appear that in the uncomplicated inflammation the pulse becomes irregular.

763. *Symptoms.*—Intense general uneasiness and jactitation are very usual symptoms; diffused tenderness of the skin has been noticed by Bright; rigors announce the onset of the disease; and Bizot insists on the significance of general acute œdema of the trunk, arms, legs, and face. Dyspnœa, it appears, may be absent, when the disease is simple; yet it is difficult to conceive, that the vessel can be inflamed to any extent without affecting the respiration: generally speaking, other affections, directly implicating the action of the lungs, co-exist. From some observations by Corrigan, it would appear that inflammation of the mouth of the aorta may induce a series of pseudo-anginal symptoms; but on the other hand, that such is not a necessary effect, is shown by Parkes's case. Pain, with sensation of heat, in the course of the vessel, felt both anteriorly and posteriorly, on the level of the lumbar spine, has occasionally proved a prominent feature. Syncopal tendency and apprehension of immediate death were noticed by Corrigan. Of the state of the urine nothing is known; yet, as we

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* Medical Times, Feb. 23, 1850.

shall presently see, this is probably a matter of considerable importance.

764. *Mode of Death.*—Chevers, who has worthily laboured on the diseases of the vessels, infers from collated cases, that death occurs in acute aortitis with extreme prostration, sharpened or bloated and livid features, cold and discoloured surface, rapid indistinct pulse, stertorous respiration, swollen extremities, and duskiness of the superficial veins; the patients die comatose, and altogether with the aspect of persons destroyed by an animal poison. There is an asthenic variety of the disease, too, in which the symptoms are adynamic from the outset.

765. *Diagnosis.*—It will probably be generally conceded that the elements of a positive diagnosis of acute aortitis are yet to be found. If narratives may be implicitly trusted to, cases occur where, supervening on other affections, acute or chronic, acute aortitis produces no obvious effect except increased irritability and distress: clearly, the diagnosis of the disease could not, under such circumstances, be ventured on. Pain, thrill, and pulsation in the course of the vessel, with arterial murmur coasting the spine, and answering in localisation neither to a murmur of the aortic nor of the mitral valves, would be the conditions, coupled with great general distress and pyrexia, most nearly warranting the diagnosis of the disease. But it is needless to point out the varieties of states that might stimulate the entire series, except the aortic murmur; and in respect of this murmur, the possibility of its depending on chronic disease, proclaims the necessity of caution.

According to Bizot, œdema of the trunk, arms, face, and lower extremities occurring acutely, without functional disturbance of any organs but those of the circulation, indicates acute aortitis. But, on the one hand, I have seen the anatomical evidences of acute aortitis, where no anasarca had occurred;* and, in like manner, Parkes's case, one of the most indubitable on record in regard of anatomical characters, proves absolutely that the disease may exist in perfection without any such œdema: while, on the other hand, Bizot has not taken into consideration the state of the kidneys in his patients. Now, in one of the three, "Bright's disease" appears to have been positively present; and in the other two we have no assurance that it was absent. Bizot's observations were made before renal diseases were either clinically or anatomically paid much attention to.

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* Campion, U. C. H., Females, vol. vi., p. 41.

766. *Prognosis.*—Aortitis may prove very rapidly fatal; it has certainly destroyed life in three or four days, when occurring in association with other less serious states. The transparence of exuded lymph furnishes the best measure of the recency of the disease. Of the prognosis, all that can be said is, that as persons die of various diseases with the evidences of chronic inflammation in the vessel, either acute aortitis sometimes fails to kill, or chronic aortitis sometimes pursues a chronic course from the outset: both propositions are probably occasionally true.

767. *Treatment.*—(*a*) Were the existence of the disease upon fair grounds even suspected, it would seem, active measures should without delay be had recourse to. Venesection or free cupping in the course of the vessel in the front of the chest and along the spine, or, if the disease have occurred in a very low state of system, dry-cupping have been held to be measures essential to safety: the real efficacy of blood-letting, however, remains unproven. Counter-irritation, by the application of a long narrow blister along the left vertebral groove (which may be used also for the purpose of applying morphia endermically), has theoretical argument in its favour; for the mass of tissues, intervening between the skin and vessel, is in all probability sufficiently great to render the effect of the blister antagonistic.

(*b*) The internal medicines, deserving of most confidence, are opium and tartarised antimony: if the excitement of the heart and vessel were very great, aconite, digitalis or acetate of lead might be simultaneously adminstered. The utility of mercury is more than questionable. Aperients and saline diaphoretics are worthy of attention as adjuvants.

In employing these and other measures the practitioner must never lose sight of the constitutional state.

CHRONIC AORTITIS.

768. Thickening of the coats of the aorta, undue vascularity of the outer membrane, unevenness, roughness, puckering, furrowing, and channelling of the inner surface of the vessel, with various alterations in its calibre, producing irregular distension and contraction, constitute unquestioned characters of chronic inflammation. White opaque cartilaginiform patches, studding the inner surface of the vessel, are likewise admitted to be the chronic representatives of the lymph-exudations of the acute disease. The relationship of saline precipitation to these patches is made matter

of dispute; the fact appearing to be, that, while the usual nidus of calcification is certainly atheroma, the white patch is the occasional seat of the change, either primarily, or secondarily to the deposit of atheroma within itself.*

769. But, serious as these conditions are anatomically, any symptoms to which they give rise, unless they have led to, or, at least, are attended by, very considerable alterations of calibre of the aorta, in the forms of obstruction, coarctation, or dilatation, prove, as a rule, obscure. For, although I cannot avoid maintaining, on clinical and *post-mortem* evidence, that marked calcification may prove the source of notable pain [759]; yet, on the other hand, that such change of structure, even on an extensive scale, may remain wholly painless, is indubitable. And, again, until the elasticity of the vessel has been very deeply impaired, it continues capable of taking its natural part—in great measure a mechanical one—in carrying on the circulation.

§ IV.—ATHEROMA AND CALCIFICATION.

770. That atheroma and calcification of the aorta, no matter how interesting they may be anatomically, possess, *per se*, but a limited amount of symptomatic importance, flows directly from what has been said in the previous section. On the other hand, destroying, as they do, the elasticity of the vessel, rendering it fragile, contributing to, or at least facilitating, the production of various aneurismal changes, and connected with two important diatheses—the fatty and the calcifying—the significance of these conditions of the vessel can scarcely be overrated in a pathological point of view. Besides, in their ultimate possible influence they are of grave clinical interest: for calcareous matter may be the occasion of obstruction, it may be obliteration, of the vessel (in its abdominal portion), by protruding more or less into the interior, and affording a nidus for the blood to coagulate around and form thrombi [840].

771. Unless such mechanical results are produced, both subjective and objective evidences of the anatomical changes are wanting. Atheroma never causes pain, as far as I know; the occasional influence of calcification in this way has already been admitted. Neither condition necessarily interferes, to any perceptible amount, with the movement of the blood-stream.

772. And of physical signs of atheroma there are none, con-

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* Products, Adventitious, Cyc. of Anat., p. 87.

cerning which we can feel sure. But calcification of the arch is attended with systolic rough murmur, either limited to the vessel, which is rare, or audible also in a less intense form at the aortic valves,—with jerking, inelastic impulse behind the sternal notch, and, occasionally, with systolic thrill both at that point and about the second right cartilage.*

773. Among the tactile characters of the pulse, fulness, hardness, regularity, jerkiness, and suddenness are the most striking. Now, these characters recall in some measure the distinctive attributes and the typical pulse of aortic reflux. And the close analogy between the two pulses may be further seen in their sphygmograms (*vide* APPENDIX): even the hooked apex of aortic insufficiency is found in the rigid pulse of atheroma and calcification. So complete, in truth, is the well-nigh identity in some cases, that Lorain† goes so far as to suggest the probablity, that the so-called typical pulse of aortic reflux really belongs to a coexistent hardened state of the general tract of the vessel.

§ V.—ANEURISM OF THE AORTA.

774. I. The term "aneurism," understood in its widest sense, may be defined as a local increase of calibre of an artery. And in this sense it has been used by some authors, while others have made attempts to restrict its application in different ways. Professional opinion is, indeed, so unsettled as to the proper application of the term, that an explanation of the meaning he, in particular, may attach to it, is called for on the part of every person employing the word.

775. Adhering, then, to the comprehensive definition above expressed, I would divide the *genus* Aneurism into the subjoined *species* and *varieties* :—

SPECIES.	VARIETIES.
Peripheric : *dilating.*	*a.* Fusiform. *b.* Globular.
Lateral : *sacculating.*	*a.* Simple. *b.* Compound. *c.* Mixed.
Interstitial : *dissecting.*	

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* Allen, U. C. H., Males, vol. xi., p. 330.

† Op. cit., p. 249. In point of fact hooked apex may exist without aortic reflux, and aortic reflux is not necessarily accompanied by decidedly hooked apex.

The anatomical constitution of these varieties of aneurism is exhibited to the eye in the subjoined Diagram.

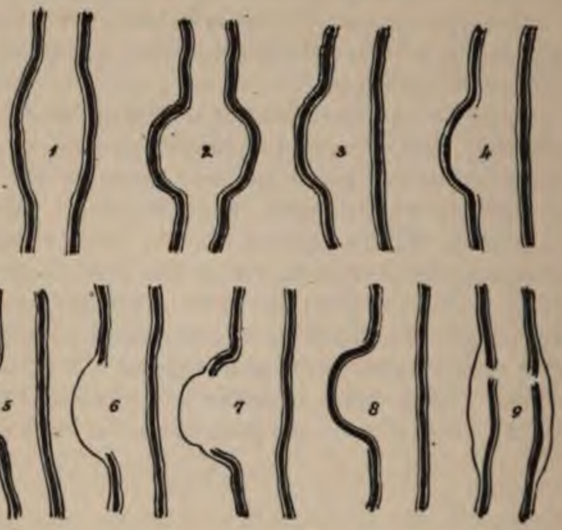

Fig. 1. Peripheric Dilating Aneurism; the three coats of the vessel all round *gradually* widening, so as to give a *fusiform* shape to the enlargement. Fig. 2. Peripheric Dilating Aneurism; the three coats of the vessel all round *abruptly* widening, so as to give a *globular* shape to the enlargement. Nos. 1 and 2 are the "simple dilatations" of various authors. Fig. 3. *Simple Lateral* Sacculating Aneurism: the sac, formed of the three coats of the vesssel unbroken, rises from a limited portion of its circumference: the "*true* aneurism" of some authors. Figs. 4, 5, 6, 8. Varieties of *Compound* Sacculating Aneurism; all agreeing in the characters of lateral sacculation, and injury to the coats of the vessel: the "false aneurism" of writers. Fig. 4. The sac formed of the middle and outer coats, the inner being destroyed. Fig. 5. The sac composed of the outer coat lined by the inner, the middle tunic having disappeared. Fig 6. The sac, composed of the outer membrane alone. Fig 8. The sac, composed of the middle and inner coats, forming a sort of hernial protrusion through the destroyed outer coat. Fig. 7. Mixed Aneurism, a combination of the simple and compound sacculating varieties; the simple condition of Fig. 3 having existed for a variable time, the middle and inner coats more or less suddenly give way, and the condition exhibited in Fig. 6 is added to the original simple disease. Fig. 9. Dissecting Aneurism; separation of the outer coat from the middle, by blood escaping from the interior of the vessel, through fissures in the lining and middle tunics.*

776. Now, in the aorta, the only vessel with which we have here to do, certain of the above varieties are distinguishable from each

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* I strongly doubt that the sac, in fig. 5, is such as it has been represented to me by authors,—namely the outer coat lined by the stretched inner membrane. The apparent inner tunic is probably nothing more than a film of adventitious formation; at all events, I have never seen a sac so constituted. Of fig. 8, there is but one positive example known, so far as I can ascertain,—namely, the preparation, No. 1642, in the Hunterian Museum. Another variety of hernial aneurism, protrusion of the inner through the middle and outer coats, has been described: in all probability imaginatively.

other by symptomatic characters, and, in reference to this important practical truth, may be placed in four *clinical* groups (differing in arrangement from the *anatomical* groups), thus:—

(Group A.) The simple and compound lateral sacculating aneurisms of figs. 3, 4, and 6.

(Group B.) The fusiform and the globular dilating aneurisms of figs. 1 and 2.*

(Group C.) The mixed aneurism of fig. 7.

(Group D.) The dissecting aneurism of fig. 9.

By far the most important of these groups is the first. Its constituent varieties in the present state of knowledge are not distinguishable by any positive signs during life; hence their association in this clinical scheme.

777. The effects, signs, and characters of aneurisms of the aorta differ so much in the chief divisions of the vessel, that the separate consideration of the disease, first, in the arch, with the ascending aorta conjoined, secondly, in the descending thoracic, and, thirdly, in the abdominal divisions, is matter of absolute necessity.

I.—ANEURISM OF THE ARCH OF THE AORTA.

GROUP A.—SIMPLE AND COMPOUND LATERAL SACCULATIONS.

778. Aneurisms, belonging to this group (figs. 3, 4, 6), affect a portion only of the circumference of the vessel; are generally narrower (the compound much more so than the simple) at the neck than at the body of the sac; rarely spring from the inferior curvature of the arch: range in size from that of a nut to that of the fœtal head; and are filled to variable amounts with laminated coagula,—the compound more extensively, as a rule, than the simple, because the orifice of its sac is comparatively narrow. The influences of these aneurisms must seriously differ, according to the precise direction in which the sac chances to enlarge; but a grand practical distinction of specimens belonging to the present group, may be established, in this point of view, into the *centrifugal* and the *centripetal*,—those tending to grow outwards towards the walls of the chest, or inwards towards deeply-seated parts. The former habitually attain much greater bulk than the latter, for the simple reason that organs of of vital importance being secure from

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* Globular dilating aneurism, apparently most common in the cerebral arteries, is very rare in the aorta. A well-marked specimen will be found in the U. C. Museum, No. 4036.

pressure, the essential functions are, comparatively speaking, but little interfered with, life is prolonged, and time given for the free enlargement of the sac.

779. The *effects of an aneurismal sac on the adjacent parts* are of various kinds: but all, directly or indirectly, due to pressure.

(*a*) Mere *displacement* is illustrated by detrusion of the trachea, or œsophagus, sidewards or backwards, of the heart downwards; by anterior bulging of the walls of the chest; by dislocation of the sternal end of the clavicle,[*] and by depression of the apex of the lung.[†]

(*b*) *Interference with the freedom of hollow canals* is seen in obstruction of the œsophagus, the trachea, either large bronchus, the descending cava, the innominate, left carotid, or left sub-clavian arteries,—vessels which, blocked up either by direct pressure, by twisting of their orifices, or by coagula, become more or less functionally disabled.

(*c*) *Injury to parenchymata* is exhibited in compression of lung-substance, condensed so as to be incapable of expansion.

(*d*) *Destructive and perforative absorption of tissue* may occur in the trachea; the œsophagus; the pleura, lung, and bronchi; the pericardium; the pulmonary artery; the substance of the lung itself; the thoracic duct; the ribs,[‡] sternum, rarely the clavicle, the vertebræ,—though aneurism of the arch seldom, however, actually makes its way into the spinal canal; the recurrent nerve, and the spinal nerves on the left side.

(*e*) *Exudation of lymph* often takes place between the aneurismal sac and the parts pressed on, as the ribs and sternum, the pleura and lung.

(*f*) *Irritation of nerves* is exhibited in various spasmodic phenomena connected with traction of the recurrent nerve.

(*g*) And, lastly, actual *inflammation of texture*, as an indirect result of pressure, is displayed in the tracheitis and bronchitis that so often accompany the disease.[§]

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[*] The disarticulated head may sometimes be distinctly felt rising from its normal site; *e. g.*, Covey, U. C. H., Males, vol. xi., p. 316.

[†] Brader, U. C. H., Females, Clin. Lect., Lancet, *loc. cit.*, p. 117. No lung-substance reached higher in this instance than the second rib on the affected side. The aneurism sprang from the descending portion of the arch, just at its union with the transverse, and was combined with small fibro-fatty tumour in both mediastina.

[‡] Small aneurisms destroy contiguous bone more readily than large ones,—obviously because pressure is more concentrated in the case of the former.

[§] For further illustrations of the effects of intra-thoracic pressure, I must, in order to avoid repetition, beg to refer the reader to the section "Pressure-Signs" in the volume on the Lungs.

(*h*) *Overgrowth of the heart*, very certainly follows in a certain share of cases, especially where the sac originates in the vicinity of the valves. But it is equally certain (the statement is made on *post-mortem*, in addition to clinical, evidence), a very large sac, of some years growth, and situated in that part of the vessel, may fail to induce the smallest increase in the heart's bulk.

Now, as will be readily understood, many of the different effects, just enumerated, are useful in guiding the observer to a *precise localisation of the disease*. But there is a peculiarity, occasionally observed in connection with these effects, which, unless carefully interpreted, may lead to grave error of diagnosis. Some one or more of the number may disappear, giving place to others: thus, well-marked œdema of the shoulder and lateral base of the neck, caused by pressure on the corresponding innominate vein, or agonising pains induced by pressure on the brachial plexus, may go wholly away, giving place to bronchial irritation, to laryngeal spasm, or to œsophagism. The explanation is simple: the direction of enlargement of the sac has changed in such manner, as to remove pressure in one spot and transfer it to another. The same kind of change, though to a notably less degree, is obvious in occasional cases of mediastinal tumour; and in the instances both of aneurism and solid growth, I have known the diagnosis resolutely contested on the ground of such change.

780.—*Physical signs of a sacculated aneurism of the arch.*—The physical signs of a sacculated aneurism of the arch are in well-marked cases extremely numerous, and as precisely and directly significant as, perhaps even more so than, in any other disease. But, on the other hand, the instances are rare, where the signs are numerously associated, unless the dilatation have attained very considerable dimensions; any one of the series of signs may be absent throughout the course of the disease; and sometimes during its earlier periods there is not a single one of the number satisfactorily developed. Hence it comes to pass, that the diagnosis of aneurism of the arch may be the easiest or the most difficult of achievements.

(*a*) *Inspection.*—By inspection, *local bulging* is discovered, tending, when at all notably prominent, to the gently conical form. The ribs and interspaces equally contribute to its formation, when the area of the bulged surface is at all extensive; but when of limited extent, that is at the time, the sac commences first to act on the surface, a single rib or cartilage may alone be prominent, or the end of the clavicle simply pushed forwards. In cases of long standing, and where the base of the conical bulging is extensive, the skin becomes smooth and glazed-looking, the unevenness depending on the ribs and interspaces, disappearing.

Concerning the seat of this bulging: first, if the sac spring from the first division of the arch, including the ascending aorta, the prominence mainly appears at the right edge of the sternum, in the second interspace and second cartilage, up to the first, and even as far sometimes as the clavicular joint. Secondly, if, while the sac involves this division of the arch, it likewise implicates the transverse and somewhat the descending portions, the prominence, instead of lying to the right, may be placed solely to the left of the sternum, in the intra-clavicular and mammary regions, extending as far outwards as the line of the nipple; * obviously the ascending part of the arch undergoes a process of twisting to the left. Thirdly, if the tranverse division be the affected part, the top of the sternum, or the right first cartilage forms the prominence; but if the sac be of small size, actually limited to this segment of the vessel, and springing from its posterior aspect, there may be, or, more correctly, will be, no visible bulging at all. Fourthly, when the descending portion of the arch is sacculated, the prominence appears at and about the second left cartilage: but aneurisms thus situated do not often cause anterior bulging; this portion of the vessel lies far away from the front of the chest, and, as matter of experience, the tendency of its aneurisms is to enlarge to the left side, while they expend their force posteriorly in eroding the vertebræ. Fifthly, the sac may protrude at the base of the neck. Whatever be its seat, the superficial extent of the prominence is less than the diameter of the sac; the bulged surface corresponds to the most prominent part of the latter. The actual area varies between the size of half-a-crown and that of a large cocoa-nut. Deficiency of bulging is rarest where the sac lies to the right; commonest, where it lies to the left; of medium rarity, where the horizontal and central portion of the arch is implicated.

Movement, pulsatile, expansile,† and synchronous with the

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* Harris, U. C. H., Males, vol. ii., p. 262. The sac in this remarkable case commenced two inches above the aortic valves, and terminated close to the left subclavian; the innominate artery was carried to the left, so as to lie in front of the origin of the left carotid.

† The expansile character of the pulsation was shown to the eye remarkably in the case of Warren, U. C. H., vol. viii., p. 25, 1849. A belladonna plaster had been placed over the aneurismal bulging. which was remarkably broad-based; as the plaster produced an uncomfortable feeling of tension at first, it was slit across hori-

heart's systole, may be seen in the bulged part of the anterior chest; and, in some cases, motion of more abrupt and non-expansile character is very perceptible to the eye above the clavicles, evidently coming from below and as evidently produced by a body of some size. But if a sac, even though of massy bulk, be filled in great measure with fibrine, while a dwindled current of blood trickles with more or less difficulty through a small channel at the furthest aspect from the anterior surface of the chest, that sac may, especially if seated in the descending part of the arch, be as absolutely pulseless to the eye, as if totally unconnected with the arterial system.

(*b*) *Palpation.*—The hand, applied to the bulged surface, appreciates more accurately, than can the eye, the motion of the sac. Generally a very little behind the apex-beat of the heart in point of time, the systolic impulse of the aneurism is sometimes, as far as the sense of touch can determine, synchronous with this. By systolic impulse, it must be remembered, is intended that synchronous with the systole of the heart: if the vessel alone were considered, this impulse should of course be called diastolic. In certain instances, by no means in all, the impulse is double, a receding as well as an expanding motion existing,—nay, in some cases of the sort, the diastolic action amounts to distinct impulse against the chest-wall, instead of, as usual, a mere receding jog. The force of the systolic impulse is sometimes extreme; even from a sac of small size, and mainly seated behind the sternum, which may have undergone but very slight erosion, the throb may be sufficiently strong to shake the head applied to the stethoscope.* I have known the diastolic impulse even similarly forcible.† In character, throbbing simply or thrusting and heaving, dull and inelastic (the latter condition marked in proportion to the quantity of fibrine in the sac), there are cases in which it is very positively undulatory. When the walls of the sac reach directly to the surface,—when they have bulged this considerably,—when there is a free passage through the sac,—and when laminated coagula in but small quantity line its interior, the current of the blood passes with wave-like motion directly beneath the finger laid on the skin.‡ The seat of palpable, is the same as

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zontally; subsequently, with each throb of the sac, the edges of the incision diverged, by about a line.

* Downie, U. C. H., Males, vol. iv., p. 273.

† ———, attending U. C. H., 1861.

‡ Harris, U. C. H., Males, *loc. cit.*

that of visible action. When the descending portion of the arch is aneurismal, the impulse of its transverse division, which we will suppose sound, may be considerably intensified, as felt by a finger placed in the sternal notch; or it may vary from day to day, independently of any concomitant change in the impulse of the prominence directly over the sac,—a circumstance sufficiently calculated to puzzle the observer as to the real seat of the disease. Aneurisms seated in this part of the arch tend also to raise its transverse division slightly above the natural level. In rare instances, very positive impulse may be detected in the inter-scapular region, more frequently on the left side than the right,—a fact explained by the relationship of the arch on the two sides to the spinal column. Sometimes unnatural impulse in the region of the arch may be discovered by laying one hand on the top of the sternum, the other in the inter-scapular region,—the single hand having failed to distinguish anything abnormal.

Very much importance has been attached by various writers to a sense of *tactile* expansion accompanying the impulse, as a distinctive sign of aneurismal pulsation. Of its occasional occurrence there can be no doubt, as, especially when under circumstances just referred to, expansion is plainly *visible*. But the practitioner must remember that tactile expansion may be wholly absent in cases of sacculation, and as positively present, where the hand really receives the impulse of a solid substance, for instance the kidney [755, *b*], lying in front of a normally sized vessel.

Systolic vibratile thrill of variable amount may attend the impulse, or be perceptible, even where distinct impulse cannot be detected. In aneurisms of the group under consideration, thrill is less constant than in the group of peripheric dilatations; but I have known it intensely marked,—sufficient almost to tickle the hand. It appears to attain its maximum in cases of lateral sacculation grafted on general dilatation. Thrill may be limited to the bulged surface, extend slightly in all directions beyond its confines, or be perceptible above the clavicles and sternal notch. The sign may be more distinct in the latter situation than over the sac itself. Thrill may exist at one time, disappear and return more forcibly than ever,—a fact in some instances explicable by the varying state of the blood in respect of anæmia, in others referrible to changes in the state of the sac, its contents, and its inlet.*

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* *E. g.*, Case of Harris, *loc. cit.*—Thrill disappeared December 4, returned December 21.

The vocal fremitus may be annulled, over a sac of medium size, under the right clavicle.[*]

(*c*) *Mensuration.*—Aneurismal sacs, according to the direction in which they point, increase the measured distance from the sternal notch to the nipple, the middle line to the nipple, or both. They interfere with the measurable chest-expansion of breathing on the surface corresponding to themselves, and may restrain this very greatly by pressure on a chief bronchus.

(*d*) *Percussion.*—Dulness, with resistance, under percussion, exists at the surface nearest an aneurismal sac. The reasons, why the superficial extent of dulness should prove, practically speaking, less than that of the aneurismal pouch and artery, from which it springs, combined, have already been explained [214]. An area of some square inches may be rendered dull by sacs of very large dimensions. The observer must always bear in mind the possibility also of the real dulness of a sac being factitiously increased in extent by adjacent consolidated lung, or solid accumulation in the mediastinum.

The seat of dulness in *front* of the chest bears the same relationship, as that of prominence, to the different portions of the arch. As a rule, it is most easily and completely detected to the right of the sternum and in connection with the ascending portion of the arch and its angle on the right side; but if twisting of the vessel take place, the deficiency of resonance, even though the ascending portion of the arch be implicated, strikes us most obviously to the left of that bone.[†] Nor can it be said that a sac springing from the descending portion of the arch, even when small, fails to effect the percussion-sound; but, for obvious topographical reasons, it does so proportionately less, both in superficial extent and in amount. A sac, originating in the horizontal part of the arch, impairs the natural osteal quality of the resonance immediately below the sternal notch, the edges of the lungs being pushed sidewards to a variable extent: it also affects the sound above and to either side of the bone. Whatever be the seat of the pouch, it is important to ascertain positively, whether the dulness connected with it does, or does not, reach completely into the acromial angle of the infra-clavicular region. Aneurisms of moderate size seated in the horizontal and descending parts of the arch give dull sound *posteriorly* in the inter-scapular regions; and a large sac, even though derived from its ascending portion, will impair the resonance between the right scapula and the spine.

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[*] Mr. ——, seen with Mr. J. Tapson, October, 1852.

[†] Harris, U. C. H., Males, *loc. cit.*

If a sac be moderately stratified internally with fibrine, the character of its resistance is nowise special. If, on the contrary, it be closely filled with such coagula, and the amount of fluid blood within it be small, the resistance is dull, inelastic, and putty-like. This kind of resistance is significant enough, when discovered; but it is scarcely necessary to add, that either for the purpose of eliciting this, or any other, character of percussion, the least roughness in manipulation is not only awkward and *unclinical*, but actually dangerous.

In a former place [214], I have touched on the question of the smallest amount of dilatation of the arch capable of being demonstrated by percussion. Under a concourse of favouring circumstances, we have seen that a very small increase of size may be so discovered. The inefficiency of percussion increases with the smallness of the sac and its distance to the left of the median-line. There can be little doubt that more than one small aneurismal sac, in the middle division of the arch, has escaped detection, simply because percussion was not performed below, and at, the sternal notch.

(*e*) *Auscultation.*—Few diseased states give rise to such variable auscultatory signs, as a sacculated aneurism of the arch,—a fact sufficiently proved by the following series of conditions of sound that have actually fallen under my notice: the list probably might be increased from the experience of others. Through the stethoscope, placed on the most prominent part of the surface, I have heard—

1. A double sound, both divisions of which are rendered murmur-like by suspension of the respiration, while both are weaker than the sounds at the base of the heart. 2. A double sound of the same characters, except that its divisions are louder than the basic sounds of the heart. 3. No sound at all, properly speaking; but a dull, impulsive impression, systolic in time, that simulates sound. 4. A systolic impulsive impression of the kind, with a pure ringing second sound.* 5. A systolic blowing murmur, harsh in quality,—harsh to the same, a greater, or a less amount than elsewhere in the artery close to the sac; with a dull muffled diastolic sound. 6. A roaring, grating systolic murmur, stronger than at the base of the heart, the aortic valves being constrictively

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* Bell, U. C. H., Males, vol. vii., p. 168.

diseased, and the blood spanæmic; with a diastolic sound. 7. A double rough murmur, the systolic division louder than the diastolic. 8. A double rough murmur, the diastolic division louder than the systolic. 9. A systolic sound, with a diastolic murmur,—a very rare combination. 10. In systolic time, sound and murmur both, and distinct from each other; in diastolic time, a loud, ringing, clanging sound.*

It is to be understood of all these varieties of aneurismal audible signs, that the influence of valvular disease in their production is, as matter of observation, supposed to be excluded.

Aneurismal sacs may then be, as it were, silent; or be the seats of sound or murmur, either singly or both combined. On the relative frequency of these different acoustic states I can make no positive assertion; nor do the elements for such assertion exist anywhere, that I am aware of. But of one fact, that murmur is vastly less frequent than the language of the majority of writers would indicate, no one, accustomed to clinical observation, entertains the least doubt. Another fact not generally known, or at least habitually lost sight of, is that the conditions of sound or murmur over a sac not only vary at different periods of the disease, but even occasionally, from day to day, in different postures, and at different phases of the respiratory act.

The *sounds* heard over aneurismal sacs are, generally speaking, identical in special character with those of healthy arteries,—they are simply more intense. But occasionally, a peculiar character in the single or double sound of an aneurismal sac, when completely free from murmurish quality, may be perceived, that seems best designated by the phrases *pumping* or *sucking:* this variety is not without diagnostic significance.*

Of the particular attributes of aneurismal *murmurs*, the following are worthy of clinical attention. The quality of the *systolic* murmur may be simply blowing, or blowing with a peculiar hoarse hollowness (which, when well marked, is important in diagnosis), grating, rasping, sawing, filing, or, if the blood be spanæmic, roaring. Variable, but generally low, in pitch, this murmur may be of higher pitch, nevertheless, than a co-existent systolic murmur at the base of the heart.* Short and abrupt in the majority, prolonged, almost drawling, in the minority of cases, its intensity may reach, or even exceed, that of the loudest murmurs produced within the heart. The strength of the *diastolic* murmur, though variable, is rarely great, absolutely speaking; in quality, it is generally softer than the systolic: it is not constantly present, not only at different periods of the same case, that has at one time presented it, but even with successive beats of the heart. Generally best audible over the most projecting point of the prominence, murmurs may, on the contrary, attain their maximum loudness at the edges of this: or, even when the main part of the aneurism lies considerably below the clavicles, they may be strongest at the base of the neck—probably, under the latter circumstances, thick layers of fibrine are accumulated in the lower parts of the sac; or, lastly, they may, in very rare cases, exhibit greatest intensity on the left side of the spinal column.

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* Moriarty, U. C. H., Males, vol. xi., p. 164. In the varieties 4 and 10 (described as in the edition of 1861) will, I think, be found the condition of the second sound, subsequently insisted on by J. W. Begbie as diagnostic of aortic aneurism, or dilatation (on Accentuated Cardiac Second Sound, Ed. Med. Journal, June, 1863). Begbie, however, claims for this condition of the sound greater diagnostic significance than had been ventured on by myself.

The mechanism of these murmurs seems to be as follows. The systolic is either produced by the passage of blood over a surface roughened by fibrinous masses, inequalities in the coats of the vessel, and calcifications; or it may come of the rippling motion given to the fluid on its entry from a portion of tube of natural calibre into one more or less dilated, especially if the dilatation be abrupt; or it may be caused by the flow of blood through the comparatively narrow and more or less rough orifice of the sac; or, not directly dependent on the aneurism itself, it may proceed from a spot of the vessel pressed upon and rendered narrow by the sac. Or it may originate beyond the sac, and arise from the passage of the blood-stream from the dilated part of the vessel into that of natural calibre.† On the other hand, diastolic murmur seems due to the reflux of blood from the sac through its orifice; and as the force of the reflux current must be comparatively slight, so, as a rule, diastolic aneurismal murmurs are feeble: sometimes, where undue force is given to the back current by coexistent aortic regurgitation, the diastolic murmur, especially if the sac be very close to the origin of the aorta, acquires unusual intensity. When the entry of the blood into the sac is murmurless, and its escape productive of murmur,—that is, when diastolic murmur alone can be heard,—the peculiarity probably depends on some special condition of the orifice of the sac, whereby a smooth surface is presented to the entering blood, and a surface, roughened by moveable fibrine or otherwise, opposed to the receding current.

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* I do not believe, however, that an aneurismal murmur is ever of, absolutely speaking, very high pitch.

† Mr. ——, seen with Mr. P. Duffy.

A number of conditions tend either to enfeeble or intensify aneurismal murmurs. If the heart's action be very weak; if the sac be filled in great measure with fibrine; if the sac be incapable of much expansion; if the orifice of communication between the sac and the artery be very large, inasmuch as the current is then too free; or if that opening be at once very narrow and smooth, inasmuch as possibly the stream is then too small to generate notable sound,—if any one of these conditions be present, a sac, otherwise well constituted for the purpose, may fail to furnish murmur. On the other hand, great roughness of surface, neighbouring pressure, and the presence of good conducting material round the sac, will intensify, really or apparently, these murmurs. Mere sounds may sometimes be rendered murmurish by suspension of the breath for a moment,—a fact to which it is not easy to supply a satisfactory clue.

A murmur may be suddenly generated in a person, known subsequently to be the subject of aneurism of the arch, be audible not only to himself, but to bystanders at some distance off, pervade the entire arterial system, though of maximum loudness at the upper part of the chest, and disappear, at least as a phenomenon audible without the aid of auscultation, mediate or immediate, as suddenly as it came.* More than one hypothesis readily suggests itself in explanation of this singular condition; but, as I have no *post-mortem* evidence, proving the reality of any one possible mechanism rather than of another, I abstain from conjecture.

I have recently met with another very similar case. A gentleman, who had no reason to doubt up to that moment his being in perfect health, suddenly, while engaged in writing, heard a loud murmur referred by himself to the chest, and proved by auscultation to originate at the aortic arch, while it was audible almost over the entire arterial system. There was no additional evidence of aneurism a week after the murmur first became audible; but of the issue of the case I unfortunately know nothing. [245.]

781. *Symptoms* (*a*).—The weight and flesh of aneurismal patients undergo very considerable reduction in a certain share of cases; while instances, where the sufferer continues stout to the

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* Facts all illustrated by the case of Mr. ——, seen with Mr. Pollard of Brompton; death occurred by rupture of the sac.

last, or almost to the last, are far from very uncommon. The centripetal or centrifugal progress of the aneurism does not always explain this difference; if, on the one hand, the greater disturbance of important functions, attached to the former mode of progress, tends to produce rapid emaciation, the protracted, though less acute, functional mischiefs endured in the latter, eventually work out the same result. Besides, these two topographical modes of advance are sometimes so mixed up, that in not a few cases it is impossible to place the disease exclusively in one or the other category. But from the analysis of seventeen cases of aneurism of the arch, of which I have notes, the inference clearly flows, that the presence or absence of pain of serious character is the real element in determining, or warding off, early emaciation. And that pain is very decidedly greater in cases where the dilated vessel bears either against the chest-wall and intercostal nerves, or the spine and vertebral groove,—hence where the course is centrifugal. It appears that extreme emaciation has sometimes been mechanically caused by pressure on the thoracic duct. The face, trunk, and limbs waste *pari passu*—the face not presenting that relative fulness often so remarkable in the emaciation of phthisis.

There is not any attitude, posture, or mode of decumbency peculiar to the subjects of aneurism of the arch, as a class and for a permanency; in bed the patient usually lies on the back, with the head moderately high. But, for the relief of particular kinds of pressure, peculiar attitudes may be assumed; thus, where a sac bears upon the trachea, the patient steadily keeps the head forwards, or forwards and sidewards,—and also frequently raises or throws back the head suddenly, keeping it in this posture for a time, so as to project the sac forwards from the windpipe.* When paroxysmal attacks of dyspnœa, from tracheal pressure or irritation of the recurrent nerve, occur, the sufferer sits up with his head supported on his hands, the elbows resting on the knees, or bends over the back of a chair, &c. The sleep is not affected by the aneurism itself; but if pressure exist, the ordinary slumbers are fitful, interrupted by starts, and frightful dreams. During the urgency of bronchitic and asthmatic seizures, the patient may pass night after night out of bed. The easiest attitude in sleep may be one, which to a healthy person would prove unbearable:

* In a case recently seen, the existence of this habit, combined with slight gnawing interscapular pain, drew my attention to the arch of the aorta, where, with much difficulty, the physical signs of a small sac were elicited.

thus, with the head supported sidewise wholly on the hand, and the trunk on the bent elbow:[*] postures of the kind may also be assumed consensually during sleep, the patient having fallen asleep in quite a different attitude.

The expression of the face varies: it may be calm and not indicative of suffering, except during paroxysms of dyspnœa, growing then, terrified and imploring; or habitually cross and irritable; or anxious and worn; or simply significant of profound distress. The differences in the original temper of patients modify their facial expression during this, as all other chronic diseases. The colour of the face may to the last remain florid in the main, with slight lividity;[†] or be habitually livid; or in no single point remarkable; or pale, sallow, and cachectic-looking: the latter appearance alone can be held in the least degree distinctive. The expression of the face may undergo singular alteration by distension of the angular, and adjacent part of the frontal, veins.[‡]

(*b*) The skin of the general surface, though sometimes sallow, cannot be said to exhibit any habitual special tint. Sweating, the reverse of an ordinary symptom, may nevertheless give considerable annoyance,—and this, where the lungs are sound. The lower extremities remain singularly free from œdema; a fact often finding its diagnostic application. Slight bulbousness of the finger-ends, confined to the side on which the supply of blood is limited through the influence of the sac, may occur.[§] Œdema of the base of the neck, the face, and the upper extremities, and one or both sides of the thorax, follows pressure on the superior cava, or one or both innominate veins. The peculiar spongy elastic fulness of the base of the neck, looking like a *collar* of flesh, due to capillary and arteriolic turgescence, is also observed. The integuments over the sac sometimes alone become œdematous, from irritation or mere distending pressure. The temperature of the arm, of which the circulation is impeded, may fall very notably below that of its fellow.[§]

(*c*) The joints are not affected; no positive connection exists between either rheumatism or gout and aneurism.

(*d*) The lips full, tumid, and livid, the tongue œdematous at the edges, and of purplish tint, the mucous membrane of the pharynx, thick, livid, and coated with viscid secretion, when other signs of venous pressure exist, display, if these be absent, no peculiarity of appearance.

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[*] King, U. C. H., Males, vol. vii., p. 89.
[†] Brader, U. C. H., Females, *loc. cit.*
[‡] Moriarty, U. C. H., Males, vol. xii., p. 3.
[§] Mack, U. C. H., Males, vol. ix., p. 210.

Dysphagia, slight or severe, felt more in some postures than in others, paroxysmal or permanent, or both combined, or in some severe cases disappearing completely for a while, after having been a constant condition (a change sometimes explicable by diminution of pressure through hæmorrhage from the sac), is a symptom of considerable frequency. More commonly attending the disease in the descending and transverse parts of the arch, dysphagia may be absent even in the former case,[*] and co-exist with a medium-sized sac springing from the right angle of the arch.[†] The intensity of dysphagia obviously depends in the main on the general and local nervous susceptibility of the patient; a slight amount of pressure on the œsophagus will produce greater difficulty of deglutition in some persons, than actual perforative destruction of the coats of the tube in another.[‡] Blood may be discharged in large quantities, from rupture of the sac, into the œsophagus,—an event which, moreover, need not prove immediately fatal. Whether slight oozing of blood may take place by filtration into the œsophagus, and be discharged by sputation, without actual hæmorrhage, I do not know from experience; but there can be no reasonable doubt of the possibility of the occurrence.

The appetite fails altogether, if the aneurism be the source of pain; commonly it is capricious. Blood in small quantities, darkened and otherwise altered by the gastric fluids, is occasionally vomited, after having trickled into the stomach from filtration through the walls of the sac and œsophagus; blood of similar origin may also be traced in the stools.[§] The bowels are habitually constipated in advanced cases, probably from the patient's inability to take exercise; flatulence distresses many, even male, patients; piles and pruritus about the anus seem to be more frequent than in the average of persons of equal age. Ascites does not occur.

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[*] Brader, U. C. H., Females, *loc. cit.*

[†] Downie, U. C. H., Males, *loc. cit.*

[‡] A perforation the size of a shilling may exist without the very least dysphagia occurring during life; though, too, the current of blood in the sac must have borne, directly almost, against the gullet; though, too, the wall of the sac may be destroyed at the spot, and portions of its fibrinous coagula protrude slightly into the interior of the œsophagus: Brader, U. C. H., Females, *loc. cit.*; I repeatedly saw this woman swallow with perfect ease. In King, too, U. C. H., Males, *loc. cit.*, a smaller perforation existed without dysphagia.

[§] I have met with one case of repeated hæmorrhage through the mouth, having all the characters of the dark variety of hæmatemesis, and in which, the lungs and stomach not being demonstrably unsound, there were several signs and symptoms of aneurism of the aortic arch.

(*e*) Painful, hoarse, clanging, laryngeal cough, laryngeal rhonchi, dry or moist, audible sometimes at a distance, and dyspnœa, at once habitual and increasing paroxysmally, coupled with various morbid states of voice, indicate deep disturbance, functional or organic, or both, of the larynx. The speaking voice may be husky, muffled, cracked, and hoarse; or simply weakened, or tremulous and variable in pitch, or actually lowered in register. The hoarse variety appears to depend on chronic laryngitis, with diminished current of air,—itself, in turn, traceable to pressure on the trachea; pressure on a main bronchus is not sufficient for the purpose. Œdema of the glottis, depending on congestion, venous or sub-inflammatory, has in some cases been found. Paralysis and atrophy of the muscles of one side of the larynx, coupled with flattening and compression of the recurrent nerve, explained extreme vocal feebleness in a case observed by R. B. Todd. Tremulousness and variation of note in speaking have been traced to pressure on, and displacement of, the trachea. Paroxysmal dysphonia is explained by irritative traction of the recurrent nerve. The trachea, chronically inflamed, where the subject of irritative compression, is tender to the touch; and stridulous breathing is in part due to these conditions.

Various forms of pain are, or may be, felt in the chest. First, immediately over the aneurismal prominence, pain may be produced by mere distension, by local pleurisy, or by irritation of the intercostal nerves. Secondly, pain in the neck and arms, down to the finger-ends, is traceable to irritation of the branches of the cervical and brachial plexuses. Thirdly, pain of a peculiar gnawing, terebrating character, constant, but increasing paroxsymally from time to time, exists at the dorsal spine, if the vertebræ are undergoing absorption. Fourthly, local pain and tenderness over the sternum, is sometimes connected with periosteitis advancing to suppuration.* The second class of pains are of shooting, piercing, or stinging character, may be brought on by the slightest movement, even that of turning in bed, and are more or less paroxysmal. The gnawing pain in the back cannot, as some persons have supposed, depend on irritation of the roots of the spinal nerves, or it would, contrary to what is the fact, radiate in the course of their branches. The tenderness of an aneurismal prominence is sometimes extreme,—and in certain cases associated with a sensation of heat, perceptible also to the hand of the observer. In addition to all these sufferings, a feeling of fulness, weight, load, tightness, and oppression is experienced within the chest, in some instances coupled with a dread of movement, lest something should be displaced by the change of posture. There are patients who suffer seriously from spasmodic contractions of the diaphragm, or sensation of constriction round the base of the chest,—the obvious results of irritation of the phrenic nerve.

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* Downie, U. C. H., Males, *loc. cit.*

The state of the respiration varies. If there be no pressure on the tubes, or irritation of these, or pressure or irritation of the vagi, recurrent, or pulmonary nerves, the breathing is calm. Where any of these conditions are present paroxysmally, the breathing undergoes temporary acceleration in proportion to their amount. Or, if they are permanent evils, the breathing is laboured, whistling, stridulous, audible at a distance, and the patient commonly points to the trachea, at the sternal notch, as the source of difficulty. The number of respirations per minute almost always exceeds the average of health more or less,—I have found it range from twenty-four to fifty-six. The pulse-respiration ratio is subject to great variation; thus, in one of the cases already referred to, the mean ratio throughout the time of observation being as 2·9 : 1, the extremes were as 5·3 : 1 and as 2·3 : 1; in this instance, the variations of broncho-laryngeal symptoms furnished a key to the rises and falls.* The chest-play is more or less confined. If a main bronchus be encroached on, or the mass of one lung diminished by pressure, the play on the corresponding side will be relatively deficient; and there may be special want of expiratory rather than of inspiratory power.† Dyspnœa is an almost invariable effect where the sac is of any bulk, although all annoyance of the kind may positively be wanting, *where the sac is even huge.*‡ In many instances the first symptom attracting the patient's atten-

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* Brader, U. C. H., Females, *loc. cit.* But this explanation will not always hold good: a ratio of 4·4 : 1 may coincide with very considerable irritation of the whole broncho-tracheal tract.—Mack, U. C. H., Males, vol ix., p. 210.

† Thus in Brader, the measurements of the chest below the fold of the mamma, gave,

	Medium.	Inspiration.	Expiration.
Right . . .	17⅛	17¼	16⅞
Left . . .	16½	16¾	16½

Hence the total play on the right side equalled three-eighths, on the left two-eighths, of an inch. The left bronchus was "distorted, and almost obliterated by pressure of the sac." Hence it appears, too, that the excess of the right over the left side, at the end of full inspiration, was half an inch; at the end of full expiration, three-eighths of an inch. These results prove that there was a deficiency of expiration on the left side. They are corroborated by the facts, that the difference between full inspiration and full expiration on the right side equalled three-eighths of an inch, on the left only a quarter of an inch; while on the left side there was no difference, on the right a difference of a quarter of an inch, between the medium state and full expiration.

‡ Harris, U. C. H., Males, *loc. cit.*

tion, and commonly increasing gradually in intensity, dyspnœa acquires, *cæteris paribus*, most intensity, when the horizontal part of the arch is aneurismal. In addition to the pulmonary causes of the symptoms enumerated above, may be mentioned pressure on the auricles, pulmonary veins, or pulmonary artery, and passing accumulation of blood in the right cavities of the heart. Dyspnœa is prone, too, to occur paroxysmally at night, from accumulation of sputa, pressure on the trachea, produced by accidental movement into such postures as throw the sac against that tube, and probably from reflex action. The act of deglutition sometimes induces a severe fit of obstructed breathing.

Cough, rarely absent, may be loud, dry, and paroxysmal,—paroxysms of the kind sometimes terminate in a syncopal state, or, after great effort, are relieved by expectoration of a thin, watery fluid. Unless under the influence of accidental inflammation of the air-tubes, there may be no expectoration at all.

Blood may be discharged through the trachea in various ways. The sac and windpipe undergoing an extensive rent, a tremendous flow of blood may take place, and kill instantaneously; or, syncope occurring after copious discharge, coagula form and plug up the opening for the time;—upwards of a quart of blood may be poured out under these circumstances without immediately fatal result; still such temporary preservation of life is a rare exception to the common issue of such ruptures. Or, lastly, the expectoration may be habitually tinged with blood, so as to produce the red-currant jelly appearance: such expectoration, which is very unusual, does not always derive this character from blood filtrating from the sac, but sometimes from pressure on the vessels of the lung.* Moderate discharge of blood, by diminishing the size of the sac, and also by diminishing congestion, sometimes affords great temporary relief of urgent symptoms. The following brief abstract of a case which fell under my observation several years ago, illustrates some remarkable points in the natural history of aneurismal hemorrhage.

Mr. R. P., June 9, 1857. Ætatis about 45, complains of obstinate pains about right chest, with a feeling as if crushed on level of right pectoralis major,—this sensation often waking him up at night. Nothing physically wrong in lungs; overaction of left ventricle, with toneless first sound: examination elicits no evidence of aneurism, carefully looked for on account of above symptoms. Dec. 7, 1857. Percussion-signs of aneurism, right second cartilage, the dulness extending across top of sternum; very slight conical-looking prominence in former site; pulsation faintly visible and palpable; no thrill; over prominence double sound, second twanging, no murmur; slight dysphagia; frequent giddiness; right radial pulse scarcely equals one-third of the left in breadth and force. January 4, 1858. The prominence has enormously increased ; chest-wall feels so thin that dare not place stethoscope on it ; radial pulses as before ; right pupil slightly larger and more sluggish than left ; occasional oppression ; very slight cough ; dysphagia as before. Rupture of the sac through the chest-wall now appearing imminent, I made arrangements with Dr. Allchin, who lived in the neighbourhood of the patient, for such measures as appeared advisable in the event of its occurring. January 26, 11·30 P.M., rupture took place ; 30 to 40 oz. of blood lost, without syncope ; gradually ceased in about three hours. Gradual rally, with occasional oozings, till Feb. 5th, when copious flow occurred, though less than the first. Feb. 9, discharge to extent of about 40 oz. From this time to March 8th some five or six bleedings occurred, varying in quantity from 2 oz. and 3 oz. to 6 oz. to 8 oz. : on this day a serious discharge to amount of from 30 oz. to 40 oz. ; from this to the 17th March frequent slight oozy discharges took place, on which day a sudden burst of blood was followed by instantaneous death, the clots resembling slightly-coloured size, and the discoloured linen of salmon tint. During all this time the first local application was never removed, consisting of lint, tow, and matico-leaves, saturated with alum and tannic acid (the two latter frequently renewed) ; ice-bags were kept constantly applied also. From Feb. 5th to the end of that month the nervous erethism of the patient was so extreme as to render him at times almost maniacal ; this state was controlled by the free use of morphia.

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* Brader's case, *loc. cit.*, p. 121.

On removing the mass of lint, &c., after death, it was found, this lay in direct contact in two places with coagula within the sac,—the openings in the chest-wall being severally about the size of a penny and a halfpenny. Two fingers introduced easily reached the aortic valves. Hence this patient must have lived for nearly two months with a gradually increasing extent of his chest-wall and aorta replaced by lint.

Certain physical signs connected with the lungs are worth attention. Where the trachea is pressed on, the supra-sternal region sinks in very deeply on inspiration ; rhoncho-respiratory fremitus, in consequence of the powerful stridor of respiration, is carried to its maximum point. Pulmonary-percussion dulness comes of various influences exercised by the sac on the substance of the lung, displacement, condensation by pressure, and collapse from obliteration of bronchial tubes. Respiration-sound may be deficient to almost suppression, from bronchial pressure, through part or the whole of one lung, while exaggerated respiration exists elsewhere. Condensed strata of lung yield high-pitched bronchial respiration ; and dry and moist bronchial rhonchi are audible. Jerking rhythm may sometimes be caused by the pulsation of a loose sac against the trachea or lung.

(*f*) The *heart* may become hypertrophous, especially if the sac originate near the sigmoid valves : but such effect is by no means constant ; I have known its size fall quite within the limits of healthy bulk under the circumstances. In some cases, the relative widths of the arterial outlets of the heart undergo perversion ; in one remarkable instance, the pulmonary orifice, when opened out, measured two and a quarter inches, while the aortic reached four and three quarters : this state of things must, in all probability, seriously increase dyspnœa [33, *c*].*

* Downie, U. C. H., Males, *loc. cit.* As a general rule, the radial pulse undergoes

Inequality of force and fulness of the radial, carotid, or subclavian pulses, at corresponding points of the two sides, occasionally exists; and invariably where the blood-current towards one side of the body only is separately affected. Aneurisms of the transverse portions of the arch on the whole best exemplify this kind of irregularity. Though other explanations have been suggested, it appears that pressure on, or obstruction at, the aortic origin of the weakly-beating vessel is the only positive cause of the difference.[*] The vessel on the affected, beats a little later, too, than on the sound, side. There is a certain sharpness and jerking character in the pulse, sometimes not unlike that of slight aortic regurgitation: but the superficial arteries do not beat visibly. The pulse is sometimes markedly bisferiens to the finger; however, observation does not justify the notion that the second wave depends on reaction of the aneurismal sac.

Any tactile differences existing in the radial pulses are translated graphically by the Sphygmograph (*vide* APPENDIX).

The veins of the chest, of one or of both upper limbs may be enlarged, full, and knotty,—sometimes so firm that they cannot be flattened by pressure; those of the arm have been known to undergo complete obliteration by thrombi.[†] I have never seen pulsation of the jugulars in these cases. The situation of the obstructed veins will guide to that of pressure, whether on the superior cava or either innominate vein alone.

(*g*) The bronchial glands are sometimes enlarged, and increase the percussion-dulness of the aneurism in the back.

(*h*) The urine frequently contains excess of urea; it is free from albumen: on the whole it is, *quoad* diagnosis, insignificant.

(*i*) The genital organs present nothing special.

(*k*) Cephalalgia, a frequent symptom, sometimes depends on the throbbing action of the arteries, and is sometimes simulated by pain in the nerves of the scalp, from pressure on the plexuses below. Partial paralysis, sensory and motor, of an arm has

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but slight diminution in strength, unless on the mechanical principles referred to in the text. Marey supposes, that this depends on the fact, that almost invariably the bulk of an aortic sac is small in proportion to the calibre of the vessel, that is, in comparison with aneurism of vessels of the limbs.

[*] Change in the pulses may, as might be anticipated, be pretty speedily effected. Thus, in Mack, U. C. H., *loc. cit.*, p. 210,—"the left radial was feeble, yet sharpish, the right so excessively feeble that it was with difficulty counted." Dr. Reynolds had seen this patient one fortnight before, and was positive no difference then existed between the two pulses. Where difference of the kind occurs, the arteries of the lower extremities should always be examined, lest a normal irregularity in the two sides be taken for the result of disease.

[†] Ferrus, Mém. Acad. Roy. de Médecine.

occurred from pressure on the brachial plexus. The intellect is habitually unaffected to the last. It is quite conceivable that obstruction of the innominate or of the left carotid, may be carried to such a point as to interfere with the nutrition of the brain and induce symptoms; but such result is, clinically, at least very rare. I have seen the available opening of the innominate reduced to the size of a crow's quill flattened, through combined twisting of the trunk and the obstruction of coagula, without any cerebral symptoms having occurred during life. Probably this absence of suffering on the part of the brain results from the slowness with which the obstruction is habitually worked out. For there is every reason to believe that, when abruptly effected, as by dissecting aneurism of the arch, grave encephalic mischief may follow such interference with the current [812, *c*].

(*l*) Paraplegia has in rare instances followed erosive destruction of the vertebræ and pressure on the spinal cord. In one remarkable case, death occurring with sudden paraplegic symptoms and indications of rupture into the chest, the sac, it was discovered, had burst into the pleura, and also into the spinal canal.

(*m*) As a rule, the organs of sense escape,—at least, they escape such amount of implication as readily attracts attention. I have, however, known deafness supervene, when some form of connection with the aneurismal disease seemed highly probable.[*] It has not occurred to me to observe positive failure of vision in cases of aneurism of the arch; but I have met with twelve well-defined examples of inequality of the pupils since I first noticed the peculiarity in 1853.[†] These cases have shown me that, when the pupils are affected, three different conditions may obtain: first, contraction on the aneurismal side may exist persistently; secondly, the inequality may only be shown by sluggishness under light on that side;[‡] thirdly, the diameter of the pupils may be variable, even within a few days,—that on the aneurismal side being now equal to, now notably, now slightly, smaller than, now larger than, the other in size.[§] The latter variations would prove that the pressure of the sac must act now paralytically, now irritatively, on the sympathetic at the root of the neck.*

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[*] Moriarty, U. C. H., Males, *loc. cit.*

[†] Mack, U. C. H., Males, vol. ix., pp. 211, 244. "April 23, 1853. Left pupil, in medium state, about $\frac{1}{8}$ inch in diameter; right not more than half the size; both are round and moderately brisk." This state held on during life, but after death—"May 24, . . . pupils round, both larger than during life; the right, that which was during life so notably the smaller, is now very distinctly the larger of the two." In this case the innominate artery was so compressed that a narrow button-hole slit alone opened into the arch, and the right radial pulse was almost imperceptible.

[‡] Hobson, U. C. H., Males, vol. xvi., p. 77, March 10, 1859.

[§] Covey, U. C. H., Males, vol. xi., pp. 318, 352: vol. xii., p. 31, 1855.

I have recently observed a case in which, along with contracted pupil, the temperature of the same side of the face was distinctly higher to the hand than on the other. Not having had a thermometer by me, and having seen the patient but once, I am unable to fix the precise amount of difference. But the fact illustrates the experimental results of Petit and Bernard on the section of the sympathetic in the neck [723, *b*].

(*n*) In addition to the neuralgic pains in various nerves from pressure, absorption of their substance is sometimes effected. Intercostal neuritis has in a few instances been detected.

782. *Course.* (*a*) *Pathological.*—In the great majority of cases the progress of aneurism is slow and insidious at first: the symptoms may, however, be suddenly developed, doubtless from some sudden increase in the bulk of an already existing and hitherto latent sac,—an increase generally connected with change from the simple to the mixed condition of the disease. The further course of events is either gradually or interruptedly progressive: occasionally aneurisms of considerable dimensions have remained latent to the last, as far as subjective symptoms were concerned,—while their physical signs had been merely those of a soft, solid mass within the chest.

Progressive advance towards spontaneous cure is an occasional, though unfortunately a very, very rare, mode of progress. Laminar deposition of fibrine continues to take place, stratum by stratum, till the entire sac is filled and its surface, next the blood-current, brought on a level with the lining membrane of the vessel. I have seen as many as four distinct sacs thus rendered practically innocuous in one and the same aorta; but no one of the four was larger than a good-sized walnut. Larger sacs, materially larger, have, however, been known to undergo this form of cure, and to be eventually converted into ossiform masses.

(*b*) *Topographical.*—Topographically considered, the course may be centripetal or centrifugal, or both combined: when aneurism combines both modes of progress, one almost always predominates in activity over the other.

783. *Mode of Death.*—There are five chief ways in which life may be destroyed in the subject of aneurism of the arch: by gra-

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* Petit noticed in 1727, not only contraction of the pupil, but also (thus anticipating Bernard by a century) increased evidence of vascularity of the side of the face on section of the sympathetic trunk in the neck. Vide Gairdner, Assoc. Med. Journal, 1855, and Ogle, Med. Chir. Trans., vol. xli., 1860.

dual asthenia and exhaustion; by the obstructive and irritative effects of pressure; by rupture and hæmorrhage; by acute intercurrent diseases; by co-existing chronic diseases.

(a) On the whole, actual death by gradual exhaustion is rare. Pain, insomnia, anorexia, wasting and low irritative fever, not unfrequently, it is true, bring the patient to the brink of the grave; but in several instances of the kind, observed by myself, the immediate cause of dissolution belonged to one of the other categories, especially rupture. In one melancholy case, which I saw some three or four times in consultation, the patient, unable to bear the anguish of intercostal and other neuralgic pains, which were fast undermining him, committed suicide [789].

(b) Whether the aneurism affect the ascending or the transverse portions of the arch, broncho-tracheal pressure and irritation rank among the most frequent causes of death.

(c) Solution of continuity in the walls of the sac may be effected by slow perforation, or sudden rupture. Perforation, with or without oozing, or slight pouring out of blood, may occur into parts of considerable importance, not only without immediately fatal results, but without any perceptible special effect; * the occurrence of a sudden rent of any size completely through the wall of the sac, no matter with what internal part an unnatural communication be thus set up, is however almost invariably fatal at once. A very large breakage of the adherent sac and chest-surface may however, as we have a moment since seen, take place with actual passing comfort from the ensuing hæmorrhage (through relief of congestion and diminution of various irritative pressures). There is no conceivable position into which fatal rupture has not occurred, as may be seen by examination of E. Crisp's valuable collection of recorded cases.† Where the ascending part of the arch has been affected, the pericardium has proved the most frequent seat of hæmorrhagic effusion. Of 138 cases, where Crisp found the mode of death stated, six only furnished examples of external rupture.

Hæmorrhagic effusion from a sac may be *retained*, as when it pours into the pericardium, pleura, mediastina, spinal canal, pulmonary artery, vena cava, or various compartments of the heart;

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* Brader, U. C. H., *loc. cit.*, into œsophagus. Whitaker, U. C. H., Females, vol. vi. p. 174, admitted immediately after violent hæmorrhage, having the characters of hæmatemesis, rallied, had three more attacks within thirty-six hours, dying instantaneously in the last. The stomach contained fluid blood 7 oz., and two massy coagula weighing 13½ and 7 oz.: the sac, occupying the descending portion of the arch, communicated with the œsophagus, by an opening as large as a half-crown.

† Diseases of the Blood Vessels. 1847.

it is *rejected*, when poured into the trachea, a bronchus, the lung-substance, the œsophagus, or through the chest-wall.

Death may also take place by rupture of the heart, or of the aorta itself on the cardiac side of the aneurism.

(d) Death by *dependent* acute intercurrent diseases, such as pneumonia, gangrene of the lung, bronchitis, pleurisy, pericarditis, is occasionally observed. Death through *independent* acute intercurrent diseases is very rare; the victims of cholera asiatica, typhoid (Peyerian) or typhus fevers, rheumatic fever, the exanthemata, acute Bright's disease, cerebral and abdominal inflammations, are scarcely ever the subjects of aortic aneurism.*

(e) Of co-existing chronic diseases, affections of the substance and valves of the heart, preceding, coincident with, or sequential to the arterial changes, are the most frequently conducive to the fatal issue: a rare example of death from phthisis now and again occurs; moderate emphysema is not a very unusual accompaniment. Diathetic diseases of all varieties are singularly uncommon: distinct evidences even of the fatty diathesis are rare, and I do not remember ever to have seen fatty cardiac metamorphosis in conjunction with aortic aneurism. Ofttimes the disease occurs in people of the most vigorous frame and constitution [784].

784. *Repulsions and Affinities.*—(*a*) In 108 cases of aneurism, Rokitansky found tubercle only five times—and always retrograde tubercle. Twelve cases of aneurism of the arch, by Greene, supply four examples of tubercle; 132 cases of aneurism of the arch, collected by Crisp, furnish two examples of death by phthisis. Stokes teaches, on the other hand, that "the morbid condition which most often accompanies aneurism is tubercle. . . . . I have often thought that there was a case deserving the name of consumptive or strumous aneurism, in which the same general morbid state, which caused deposition of tubercle in the lung, simultaneously affected the coats of the aorta." According, then, to Rokitansky, tubercle and aneurism are absolutely antagonistic; according to Stokes, of affinity so close that, in some cases at least, one and the same diathesis simultaneously generates both.

In sixteen cases of fatal aneurism of the arch, examined by myself *post-mortem*, one rather doubtful example of tubercle occurs, in a male aged fifty-one; in one other male, aged thirty-two, there were pulmonary cavities, and growing tubercle [786]; in all the other cases, the absence of crude tubercle or grey granulation is affirmed in my notes. On the other hand, I do not remember, in the vast number of phthisical people (and destroyed by phthisis) I have opened, to have met with any instance of progressive aneurism. Hence my contingent of experience supports the inference of Rokitansky as to the rare association of the two diseases. But the disparity of the ages most apt for the generation of tubercle and aneurism must not be forgotten; the mean age in my aneurismal cases was forty-two; and it may be calculated that, omitting children, about 63 per 100 of tuberculous adults, 13·5 only per 100 of aneurismal people, are under thirty years of age.

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* Lecture on the Influence of pre-existing State of Health on Disease. Lancet, vol. i. 1871.

(*b*) The syphilitic cachexia and gouty diathesis are said to have some connection with aneurism. As far as my own observation goes, it supports the notion that, in its tertiary and ultimate developments, syphilis may lay the groundwork of aneurism, as it apparently does of valvular disease also [566]; so that a certain attraction might be held to exist between the two affections,—but I have no satisfactory statistical evidence to adduce. It has been asserted that mercurial dyscrasia promotes the occurrence of aneurism.

(*c*) A parallel, which I cannot help thinking somewhat fanciful, has been drawn between aneurism and cancer, in regard of their constitutional conditions and effects. Like the cancerous, it is said, the "aneurismal diathesis" is never extinguished: what comes, then, of the cures, spontaneous and by art, of the disease? Frequently, we are assured, many of the arteries are involved in the same person; the assurance is directly at variance with statistical returns. And besides, admitting the fact, (after all, it is only a question of degree, for unquestionably several arteries are sometimes affected in the same individual), the analogy fails; cancers multiply through the blood; aneurism could only do so through local changes in the vessels. The aspect of the patient and the general decay of the organism resemble those observed in cancer, according to this argument: the statement seems decidedly inapplicable to the majority of cases of aneurism, and only true of those attended with an unusual share of suffering. And, further, in their mode of distribution to the two sexes, aneurism and cancer are well nigh the antipodes of each other.

785. *Diagnosis.*—The diagnosis of a sacculated aneurism of the arch, may be, on the one hand, one of the easiest, or, on the other, one of the most difficult, problems occurring in the practice of our art,—so difficult, that certainty proves unattainable, and surmise, more or less strong, alone permissible. So true is this, that it

may be well to consider apart the grounds of *objective and positive*, and of *subjective and tentative* diagnosis.

(*a*) *Positive diagnosis.*—Locally considered, the diagnosis of sacculated aneurism of the arch turns essentially on the co-existence of (1) *a pulsating prominence*, visible and palpable, limited in area, and corresponding in seat to that portion of the vessel; (2) the *signs of internal pressure;* (3) *certain arterial murmurs and sounds;* and (4) *limited percussion-dulness in the course of the arch.*

Unfortunately not one of the series is peculiar to aneurism. Thus (1) a pulsating prominence may also be produced by a small solid mass lying over and receiving the impulse of a sound aorta, —the pulsation of a mass of the sort may even be distinctly expansile to the feel [755]; or it may be due to an abscess in the mediastinum; or to pulsating empyema; or to a tumour pulsating interstitially. (2) The signs of internal pressure may be produced by solid tumour. (3) A double hoarse murmur over the arch, inaudible or only faintly audible at the heart,—a diastolic murmur similarly localised, and a pumping or sucking character of the aortic sounds, without murmur, are the most significant auscultatory signs; but it would be rash to affirm that the association of calcification of the arch with tumour, pressing on the outer surface of the vessel, might not generate auscultation-signs of deceptively similar characters. It consequently becomes necessary to pass in review, one by one, the different affections which possess certain characters in common with aneurism, and show how these, while simulating that disease, fail to prove its existence. (4) Several morbid states of the mediastina, bronchial glands, lungs, pleura, and even of the chest-wall, may accidentally render the percussion-sound dull in the course of the arch.

(α) *Tuberculous consolidation* of one apex, especially the left, with murmur in the subclavian or pulmonary artery, is distinguished from aneurism by the non-extension of percussion-dulness across the middle line; by its extension, on the contrary, to the acromial angle; by the existence of some amount of tone in the percussion-sound, and some share of resilience in the wall of the chest; by the want of abrupt and sharply circumscribed definition of the percussion dulness; and by the absence of pressure-signs, eccentric or concentric. The symptoms, local and general, are also, in the main, widely different.

(β) *Fluid in the pericardium* is distinguished by the pyramidal form of its dulness, which aneurism never simulates except under the very rare accidental circumstances already referred to [77, *c*].

(γ) In a case of *enlarged heart*, there is but one centre of motion —in aneurism two; itself and the heart. When well-defined, this is a most valuable aid in diagnosis; but it must not be forgotten, that the aneurismal sac and the heart itself may be so closely contiguous, as to render it impossible to isolate them satisfactorily. Under these circumstances it may, or may not, be a matter of ease to detect such difference in the precise rhythm of pulsation at two given spots, as to settle the question in favour of a double source of motion. But we must remember that the sac may be not only non-expansile, but actually quiescent; under these circumstances the site of the percussion-dulness and the existence of pressure-signs must be appealed to for the distinction on the side of aneurism, and a greater or less contingent of its special signs sought for on the side of enlarged heart. If general dropsy exist, this is in favour of heart-disease, positively hostile to the existence of aneurism as the sole affection.

(δ) *Intra-pleural pulsating empyema*, with its throbbing prominence near the edge of the upper bone of the sternum, simulates aneurism closely,—so closely that in the first example of the affection, that fell under my notice, the existence of aortic aneurism had been accepted as a demonstrated fact. The distinctive marks would be these, in the case of empyema: the absence of arterial sound or murmur on the prominence; weakness of the heart's sounds at the spot; absence of thrill; absence of impulse above the clavicles; equality of the radial pulses; and the total deficiency of signs of centripetal pressure, venous, trachæo-bronchial or œsophageal.

(ϵ) *A chronic sub-periosteal abscess of the sternum*, forming a small prominence in the line of the transverse portion of the arch, fell under my notice some time ago,* sufficiently resembling an aneurismal sac. But there was no impulse; gentle percussion immediately round the prominence gave normal resonance; there was no murmur, and concentric pressure-signs were totally absent. Still there might accidentally have been impulsive action of the vessel beneath, and excess of mediastinal fat might have rendered the percussion-sound dull; under such circumstances the diagnosis would have been excessively difficult.

(ζ) *Infiltrated cancer of the lung* causes retraction of the side, produces no local prominence, deepens the intercostal spaces, and frequently renders the percussion-sound tubular in the infra-clavicular region; it does not produce pressure-signs, and may be the seat of the signs of softening and excavation.

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* Marg. Motlee, U. C. H., Females, vol. v., p. 36.

(η) *Pulmonary syphiloma*, seated within, what may be called, the aneurismal area, may prove very difficult of distinction from aneurism,—receiving, as it does, impulse from the contiguous arch, and being occasionally associated with severe periosteal pains of syphilitic origin. The failure of mercurial remedies to relieve, would depose in favour of aneurism.

(θ) *Tumour* in the anterior mediastinum presents the greatest number of positive points of similarity to aneurism. Now, if there be highly-marked pulsation, a broad-based prominence with conical elevation in the centre, the murmurs most distinctive of aneurism, and a sensation of the flow of liquid beneath the integuments, there can be no doubt that, whatever other grounds for diagnosing tumour may exist, aneurism is really present. But every one of these signs may in cases of aneurism be absent: then, observe how like the two things are—a sac filled with fibrine and a solid tumour. In truth, one is a tumour *inside*, the other *outside* the arch: and obstruction from without may have the same disturbing effect, as from within, on the blood-stream flowing in its interior. Common to the two are dulness and non-resilience, usually extending across the middle line,—all the signs of centripetal, and almost all those of centrifugal, intrathoracic pressure. Under such circumstances, the question becomes one of pure probabilities. The conditions in favour of aneurism would be these: situation in the course of the arch; vibratile thrill above or below the clavicle; marked accentuation of the second sound over the prominence; gradually increasing nearness of pulsation to the surface;[*] double impulse, especially with doubling of diastolic share of impulse; dysphagia; great pain, especially of the dorsal spine; absence of œdema of the arm and chest. The circumstances in favour of tumour, and against aneurism, would be the facts of the patient being a female[†] and under twenty-five years of age; great superficial extent of percussion-dulness, especially if there were no marked attenuation of the walls of the chest; absence of any heaving motion in the affected spot; want of accordance between the sites of maximum-dulness and of pulsation; distension of the superficial thoracic veins, and the existence of currant-jelly expectoration, which, common with tumour, is very rare with aneurism.

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[*] Yet, from stratification of fibrine, the pulsation of an aneurism may grow deeper; and, on the other hand, that of a tumour may become more superficial.

[†] But this is of little value, for the excess of aneurism of the arch in males is by no means so great as that of aneurism of all arteries indiscriminately.

It is a curious fact, that where a *quiescent aneurismal sac* and a *tumour* co-exist, the usually essential sign of aneurism, namely pulsation, apparently expansile, may be furnished by the tumour; while the usually essential sign of tumour, dead, toneless dulness under percussion, unaccompanied by intrinsic pulsation, may be caused by the aneurism. This statement is well illustrated by the case of Brader (*loc. cit.*).

(ι) The means of distinguishing *coarctation of the arch* will be found with the description of that disease.

(κ) *Aneurism of the innominate artery* is distinguished by the higher position of its pulsating prominence behind or above the inner part of the clavicle; prominence appears relatively early; dysphagia, tracheal pressure-symptoms and dyspnœa are comparatively rare; the clavicle is often pushed from its place; paralytic symptoms in the right arm are greatly more frequent; the respiration-sounds are seldom enfeebled,—if they are so, the right lung suffers. In an elaborate essay on aneurism of the innominate artery, by Holland,* it is stated, that the arteries in the right side of the neck and head and in the right arm generally pulsate less strongly than on the other side,—whereas the reverse is the general fact in cases of aneurism of the transverse portion of the arch.† The same writer notes that pressure on the right subclavian and carotid diminishes or stops the pulsations of an innominate sac,—while, brought to bear on either the right or left arteries, it exercises no influence on aortic aneurismal action.

(*b*) *Hypothetical diagnosis.*—But the major difficulty arises when, from smallness of the sac and peculiarity of site, not a single physical sign of aneurism can be made matter of demonstration; and when, in point of fact, a surmise only, more or less strong, it is true, can be formed as to the nature of the disease. The evidence, such as it is, can only be worked out by a system of negation and *per viam exclusionis.*

Thus, the absence of symptoms and signs, indicative of ordinary affections of the heart and lungs in a middle-aged male suffering from persistent anomalous disturbances within the chest, even though he does not, or rather because he does not, exhibit any failure of general health, affords fair motive for suspecting aneurism. If one of these disturbances be abiding intra-thoracic pain (especially if associated with surface tenderness), the grounds for such suspicion are greatly strengthened. And if a middle-aged male (more especially one, who has suffered from a combination of symptoms, such as this), is suddenly seized with hæmoptysis, more or less copious, or with paroxysmal respiratory or vocal signs, signifying interference with the recurrent nerve, the diagnosis of aneurism, though unsustained by a single positive physical sign, would rarely prove erroneous.

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* Dublin Journal, 1852.

† This rule is, however, open to numerous exceptions; in my own cases of ascending and transverse and aortic aneurism combined, the weakly acting radial was more frequently the right

Nay more, male adults will occasionally present themselves, exhibiting all the outward signs of robust health, who energetically proclaim they "never had a day's illness," and in whom nothing can be discovered amiss except the occurrence of severe paroxysmal dyspnœa (neither asthmatic, cardiac, nor hæmic)—dyspnœal signs which stand as the sole indices of an aneurism pressing on the recurrent, and destroying life eventually, after more or less pronounced physical evidence of the disease has been obtained.

Further, the mere existence of intra-thoracic pain, if severe, whether it be paroxysmal or constant, provided the existence of any acknowledged cause for such pain cannot be demonstrated, justifies a strong suspicion of the existence of aneurism.

The sphygmograph may or may not lend aid in helping the observer through various of these diagnostic difficulties; its utility will depend on the site of the sac (*vide* APPENDIX).

786. *Duration and prognosis of aneurism of the arch and descending thoracic aorta.*—There is an unfortunate deficiency of materials on any large scale for determining the mean clinical duration of the disease. That, once developed, it will eventually destroy life is an inference, practically speaking, scarcely shaken by the few recorded examples of alleged cure. But, it is astonishing, how long life may be prolonged, even with tolerable comfort to the individual, by such simple management, hygienic and medicinal, as common sense and experience dictate. I shall not easily forget the case of a man first seen in November, 1846, with a large aneurismal prominence, pulsating so liquidly, if I may use the word, and so directly under the skin, that it became necessary to apply a mechanical protection against the danger of rupture from a blow or even from sharp friction,—a man worn with suffering,—unable to sit, lie, or stand, from pain and general uneasiness,—and wasted considerably in flesh and strength: and yet the

death of this patient did not take place till July, 1849,—he having, meanwhile, though actively phthisical also, lost the major part of his more serious sufferings, and, for a time, in a certain subdued fashion, actually enjoyed existence.[*]

It is positive that the mean duration proves greater, when the sac grows centrifugally than centripetally.

GROUP B.—PERIPHERIC DILATING ANEURISMS, FUSIFORM AND GLOBULAR.

787. (*a*) *Fusiform dilatations* (Diagram, fig. 1).—The peculiarities of these aneurisms, as compared with the sacculated, are: diffuseness of pulsation above and below the clavicle, visible and palpable; comparatively less impulse below than above, though even there, if anæmia exist, it may be very considerable; more thrill above, less below those bones;[†] rough, prolonged, rasping, whizzing or whirring murmur, systolic only, audible along the arch, and louder there than at the aortic valves, if these also be the seat of murmur. Centripetal and centrifugal pressure-signs are almost or completely wanting.

788. Anæmia and nervous excitement, by causing violent throbbing action, may simulate such dilatation; but the results of percussion will distinguish the cases.

789. (*b*) *Globular dilatation* (Diagram, fig. 2).—Examples of globular dilatation of the arch are so rare, that a note of the principal phenomena, observed in the only case I have seen, will not be misplaced:—

Mr. ——, æt. 50, January 13, 1852; intense pain, upper sternal and left infra-clavicular region, radiating to axilla and elsewhere variously; no murmur over heart, nor aorta in front; distinct systolic short superficial weak murmur in course of thoracic aorta, just at the level of the inferior angle of the scapula; no prominence or impulse visible in infra-clavicular region; no thrill above or below the clavicles; percussion dull in left inter-scapular region, and slightly so even in the right, also about first and second left costal cartilages in front, but patient so sensitive that careful percussion impossible. Respiration in left inter-scapular region, and below this peculiarly jerking,[‡] of very sharp blowing quality. March 31; impulse now visible, as also very slight general prominence about the top of the sternum, and the two upper left costal cartilages; no thrill; no murmur in heart or aneurism anteriorly; double sound over prominence, both divisions louder than at the heart. April 26; prominence more obvious, and area of dulness increased; absence of thrill or murmur, as previously.

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[*] Harris, U. C. H., Males, vols. ii. and viii.

[†] But thrill may be totally absent in this form of dilatation, as proved *post mortem*; *e. g.*, Fullaway, U. C. H., Males, vol. xvii., 1860.

[‡] The sac, being exceedingly loose, probably caused the jerking rhythm of the respiration by pulsating against the main bronchus.

The patient destroyed himself a few days after the last interview, all the measures employed (hypodermic injection was then unknown), failing to relieve his pain. The aneurismal dilatation was of large size (U. C. Mus., No. 4036).

GROUP C.—MIXED ANEURISMS.

790. The sudden extension of dulness in the situation of a *simple* sac, coupled with similar increase of pressure-signs, especially if these conditions follow effort of any kind, makes it probable that the inner and middle coats have given away, and the outer undergone additional pouching.

791. In the signs of a mixed aneurism, when actually developed, there is nothing special.

792. The gravity of the case is greatly increased through the yielding of the inner coats, and *pro tanto* the prognosis rendered yet more serious.

II.—ANEURISM OF THE DESCENDING AORTA.

793. *Physical signs.*—The signs of an aneurism seated between the termination of the arch of the aorta and the diaphragm will, of course, vary somewhat with the precise portion of the vessel affected.

(*a*) If the sac be not of large dimensions, little is to be learned by inspection; however, in certain positions, in consequence of its lying behind the heart and pushing this organ directly forwards against the ribs or sidewards, the maximum cardiac impulse may be transferred from the apex to the base, and so-called diastolic impulse also produced.

(*b*) Posteriorly the hand may detect slight arching to the left of the spine; if the arched surface be the seat of the least impulsive action, the sign becomes one of importance; but arching may be totally deficient, even though the sac be large.*

(*c*) Dulness under percussion, limited to the same situation, and inexplicable by the condition of the lung, heart or pleura, would, of course, strengthen the inference drawn from the previous sources.

(*d*) An aneurism in this situation may supply the varieties of murmur and sound already enumerated; murmurs must be stronger over the sac than over the heart, to have any diagnostic value.

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* Moriarty, U. C. H., Males, *loc. cit.*

(*e*) Feebleness or deficiency of respiration close to the spine, or over the side generally from pressure upon the main bronchus, will corroborate the evidence of the other signs.

A case, observed some while since, revealed to me the unexpected fact that every one of the physical signs of aneurism of the descending thoracic aorta may exist to the *right*, instead of to the *left*, of the spinal column posteriorly. In this singular case, marked pulsation, percussion-dulness, and a number of other signs as well as symptoms (free hæmorrhage among the number), pointed to the existence of a large aneurismal sac. Yet, so complete was the nullity of physical signs to the left of the spine, that, although well aware of the possibility of vessels becoming twisted out of their normal line and site by the growth of an aneurismal sac,* I could not bring myself to diagnose that affection with positiveness,—vacillating between it and a carcinomatous tumour receiving the impulse of the vessel. On *post-mortem* examination a sac of considerable size was found, lying to the right of the spine, and having actually dragged the trunk of the vessel across the middle line in the process of its own enlargement.†

794. *Symptoms.*—Vertebral gnawing pain and intercostal neuralgia are occasionally observed. There is sometimes a subjective sense of throbbing action, which is with difficulty distinguished from that of the heart.

The laryngeal system commonly escapes. This habitual freedom obviously depends on non-implication of the recurrent nerve, and on the mere distance of the larynx from the locality of the disease. Sometimes, however, the larynx suffers through extension of irritation from a main bronchus; the trachea, indeed, has, in rare instances, undergone direct pressure from very large sized sacs. Perforation of the main bronchus, dysphagia from mechanical obstruction of the œsophagus, or, where the disease occupies the immediate vicinity of the cardia, various gastric symptoms, simulating internal obstruction of that orifice of the stomach, have been noticed.

795. *Mode of death.*—Death occurs more frequently, perhaps, from rupture into the œsophagus than from any other single cause; curiously enough, rupture into the right occurs almost, if

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* This is far from being very uncommon in aneurisms of the arch of the aorta in front.

† Case seen once, within a few days of the fatal termination by hæmorrhage, with W. Allchin. The thoracic aorta, but without the spine, is now in University College Museum, No. 4602.

not quite, as frequently as into the left pleura. The patient dies, worn out by the effects of bronchial and tracheal pressure in some rare cases.

796. *Causes.*—Of special causes for the disease in this particular situation nothing is known. It is noteworthy that aneurism of this division of the aorta is singularly rare in females.

797. *Diagnosis.*—The affection with which an aneurism, thus seated, may most readily be confounded, is hypertrophy of the heart; the site of the strong systolic and diastolic impulse may readily deceive. But careful employment of all the methods of physical diagnosis will prevent error in well-marked cases; while it must be confessed that very small sacs behind the heart, unless some accidental circumstances throw light on their existence, are exceedingly difficult of positive detection. The obscurity, occasionally arising from displacement of the sac and connected vessel to the right, must not be forgotten.

798. *Treatment.*—The plans applicable in the treatment of aneurisms, whether situated in the arch or in the descending thoracic aorta, are so closely similar, that the two portions of the vessel may be considered together.

In proceeding to the treatment of an aneurism of the aorta, our first duty is to determine, if possible, whether we have a fusiform or globular dilating aneurism, or one of the sacculated varieties, to deal with. For, whilst coagulation of the blood within the aneurism is scarcely obtainable in the former cases, and if obtained will not effect their cure; in the latter, there is a natural tendency to such coagulation, which does actually promote, nay, sometimes effect, anatomical cure. Hence it appears that the clinical distinction of these kinds of aneurism is not a piece of mere scholastic refinement, as it has been slightingly called, but a matter of practical importance.

There is another question worthy of serious consideration. With what hope of achieving a cure do we undertake the treatment of the case? If, as experience amply shows, existing methods have failed except in the very rarest instances to accomplish removal of the disease, clinically understood, risk, even the slightest, of doing mischief to the constitution by the energetic employment of any one of those methods, should obviously be avoided.

799. *Medical Treatment.* (*a*) In cases of *fusiform and globular dilatation*, the indications are the prevention of enlargement and

rupture of the aneurism. These ends may be best secured by occasional leeching over the affected part, especially if there be local tenderness,—much better than by small bleedings from the arm. Full and repeated venesection, on the plan of Valsalva, is in this species of aneurism even less permissible than in the sacculated varieties: but a single abstraction of some four to eight ounces of blood at the commencement of treatment, more especially in plethoric persons, sometimes gives considerable relief to distressing symptoms within the thorax,—even this should be cautiously done, however, if the system at large has at all suffered from the disease. Purgatives, to such amount as to maintain a tolerably constant free action from the bowels, both from their sedative effect on the circulation, and from their preventing the necessity for effort in defecation, are essential. Diuretics do service by preventing the water in the blood from rising above par. Direct sedatives of cardio-vascular action, veratrum viride, aconite, hydrocyanic acid, and belladonna, internally and externally, lessen the violent pulsatile action of the diseased vessel, and may be cautiously, and more or less steadily, given in various combinations.

The diet should be so arranged as to support, without exciting or over-nourishing: but anæmia is even more baneful than plethora, and the starvation system must be studiously avoided. The patient should abstain from all excitement, mental and emotional, pass the greater part of his time in perfect rest, and take but moderate daily exercise on foot: carriage exercise on smooth ground may be permitted to any amount desired.

(*b*) In the instance of a *sacculated* aneurism, the object being to promote coagulation, occasional venesection, with the view of lessening the force of the current, is commonly recommended. But there is a double danger to avoid here: if too much blood be drawn, the action of the circulating system will be excited, instead of tranquillised: and if the quality of the blood be seriously impoverished, the softness of the coagulum will probably render it comparatively useless as a support to the distended walls of the vessel. Theory, in truth, does not, at the present day support the ideas of Valsalva concerning abundant depletion; and, since the diagnosis of aneurism has become somewhat positive, cures obtained by his system have ceased to be heard of. True, the recommendations of Valsalva are rarely, if ever, executed to the letter: the courage of patient and physician generally fails—in time, probably, to prevent irremediable mischief. Moderate leeching over the sac, from time to time, generally proves useful in relieving pain and anomalous distressing sensations.

The various cardiac sedatives promote coagulation by enfeebling and slackening the current; and if there be no contra-indication in the state of the heart, a fair trial of them should never be omitted. Some practitioners have much confidence in the acetate of lead. Purgatives and diuretics are useful on the principles a moment since referred to; the latter especially, because, while they diminish the water, they exercise no influence on the fibrine, of the blood.

Tannic and gallic acids, combined with digitalis, aconite, or belladonna, have appeared to me to exercise a very beneficial effect in promoting coagulation: gallic acid may be given in doses of three, five, to ten grains twice or thrice daily, with occasional intermissions, for a length of time,—its constipating effects (if such really arise; for my own part I have not observed such), being obviated by occasional doses of castor oil.

If there be the smallest amount of anæmia, iron in some one of its astringent forms, especially the perchloride, becomes a *sine quâ non* in the management of the case: even where positive signs of anæmia are undiscoverable, iron seems distinctly to promote coagulation. It should be given in association with aconite.

The bromides of potassium and ammonium give subsidiary help by tranquillising nervous irritability.

There seems little *à priori* probability, a specific remedy for aortic aneurism will ever be found; and yet the very favourable results obtained through free use of the *iodide of potassium* show that the march of time and accident may by possibility place in the hands of the practitioner of the future some agent or other deserving the title. Not only has relief of the neuralgic pains and of the general distress followed the administration of the iodide, but the local pressure-symptoms have been mitigated, and firm thrombosis has taken place within the sac, while the area of pulsation and of percussion-dulness have exhibited sensible reduction. All this in such manner, that the patient has occasionally believed himself almost or perfectly well.

The medicine should be continued for weeks or months, with occasional intermissions when evidences of iodism make their appearance. The dose may range between ten and thirty grains thrice or twice daily: a patient from the Continent came under my notice some while ago, who had been for several weeks taking the enormous quantity of one hundred and twenty grains daily in divided doses. The exceeding toleration of the drug, frequently witnessed in aneurismal sufferers, is far from being the least singular fact connected with its mode of influence.

It seemed to me at first probable, or at least possible, that the effects of the iodide might be held to indicate syphilitic dyscrasia, as playing a part in the disease. Subsequent experience, having brought a few cases before me, where, every attainable surety existed, that the patient had never had either congenital or acquired syphilis, proved that view to be untenable. Whether the drug acts by increasing the coagulability of the blood,* or by influencing the vessel-walls or the nerves, remains open to inquiry.

The influence of iodide of potassium on the progress of aneurism appears to have been discovered not only empirically, but by the merest hazard,—in this point of view the story of the acquisition of all our really valuable medicines is simply repeated. The earliest notice of the subject, I have succeeded in finding, bears the signature of Bouillaud.† The narrative, however, contains internal evidence the drug had been previously employed: and, it would seem, the accidental circumstance of a Polish patient falling under the notice of Nélaton, who declared he had derived great benefit from the iodide at Warsaw, earliest and originally led that surgeon to employ it with, as it proved, the remarkable result of causing an aneurismal tumour to disappear almost completely.‡ Chuckurbutty's original and independent advocacy of the iodide was also the result of its chance administration to an aneurismal patient with the view of relieving some totally different condition.

Cold poultices of linseed meal and vinegar, or of conium and digitalis, relieve local suffering, and probably promote coagulation.§ Ice to the surface is grateful to some patients, unbearable by others: it can rarely be kept applied for a sufficient length of time to modify the circulation beneath. Cold poultices of oak bark have appeared to me useful. If there be excessive pain over the sac, accompanied with evidences of pleural irritation, the application of a small blister close to the spot, followed by the endermic use of morphia, will soothe greatly; if there be no such irritation, hypodermic injection will prove more effectual. Chloroform, as a local appliance, sometimes relieves pain greatly.

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* Chuckurbutty, Brit. Med. Journal, July, 1862.

† Gazette des Hôpitaux, 8 Feb., 1859.

‡ W. Roberts, Brit. Med. Journal, January, 1863. I have not been able to find Nélaton's original paper.

§ But the temperature must not be too low; coagulation is prevented by cold = 40° Fahr.

Counter-irritation, even at some distance from the sac, will occasionally, in great measure, remove local suffering: thus, interscapular pain sometimes disappears, even wholly, under the application of caustic iodine between the shoulders, though the sac essentially bears forwards, and *post-mortem* examination eventually proves the vertebræ have not been eroded.

The theoretical necessity for fibrine of good quality being clear in these cases, such diet is advisable, as seems to promote its formation. However, the dangers of plethora, especially as much exercise cannot, under any conceivable circumstances, be permitted, must be held constantly in view, and over-nourishment avoided. Fluid in any quantity is injurious, and stimulants seriously baneful.

Exercise, there can be no doubt, is to be avoided as pregnant with the utmost risk. No fair argument in favour of exercise appears to me to flow from those exceptional cases, in which violent physical exertion is borne, at the moment, with impunity. Thus, it is well known that the celebrated London surgeon, carried off in 1847, by aneurism of the transverse portion of the arch, succeeded, some time after he had lost quarts of blood from the sac, and while already seriously distressed by tracheal pressure-symptoms, in performing feats of personal prowess, pedestrian and other, not only without immediate ill results, but, as he himself maintained, with relief to his sufferings. Yet the ultimate and real effect of such strain upon the circulation must have been to hasten the enlargement of the sac. On the contrary, quietness as perfect as possible is a *sine quâ non* in treatment. I have even known considerable relief to local pain, nay, even diminution of pulsation, follow the adoption, as persistently as possible, day after day and week after week, of some one particular attitude. This attitude has, and has not been, the recumbent: the patient should always be allowed to follow his instincts in finding out the easiest position; and, if this be an awkward one, he should be relieved by appropriate mechanical aid. Mere position would do vast good to aneurismal sufferers, could they only be persuaded to submit steadily and abidingly to the annoyance of such quietude: I base this statement on its favourable effects in the few cases, that happen to have fallen under my notice, in which the patient had the courage to undergo the necessary restraint.

If the aneurismal sac be very superficial, and its wall ill-protected by fibrine near the parietes of the chest, it may be necessary to apply a shield, fitted to the part, to guard it from the chances of external violence.

800. *Surgical Treatment.*—Surgery comes to the aid of sufferers from intrathoracic aneurism with both *curative* and *palliative* intentions. It is scarcely necessary to say, that the error will not be committed in these pages of treading on the province of the surgeon by an attempt to describe the procedures belonging specially to his branch of our art; but it seems indispensable a brief outline should be given of the really remarkable achievements of surgery in this department, and an opinion expressed, from the medical point of view, as to their utility.

(*a*) *Curative.*—(1) The *hypodermic injection of the watery extract of secale cornutum*, diluted with three times the quantity of rectified spirit and glycerine, has been employed with such singular success by Langenbeck, in cases of external aneurism, that the plan seems deserving of trial in the aortic disease. The quantity injected at a time ranged from half a grain to six grains. A saccular aneurism of the radial artery, about the size of a hazel nut, is said to have "disappeared completely from a single injection of two grains and a half of the extract." [*]

(2) In November, 1863, C. H. Moore performed the bold operation of introducing twenty-six yards of fine iron wire, through a canula, into a sacculated aneurism of the ascending aorta, with the design of producing local thrombosis. The operation gave no pain, and its "immediate effects were a reduction of the pulse from 116 to 92, an almost complete cessation of the pulsation in the tumour, and a diminution in its size." [†] Death, however, followed in somewhat less than five days after the operation, from parietal inflammation, pericarditis, and renal (probably embolic) exudation.

The record scarcely encourages imitation of this enterprising procedure: still the well-ordered argument of the operator, who "hopes for eventual success," deserves careful consideration.[‡]

(3) The production of artificial thrombosis by introducing a few needles into the sac (a plan greatly less venturesome in all aspects, except the embolic, than that just referred to), has occasionally been attempted with results worthy of attention. In a case of aneurism of the arch, operated on by John Marshall at University

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[*] New Syd. Soc. Retrospect, 1871, p. 278.

[†] C. H. Moore and C. Murchison, M.D., Med. Chir. Trans., vol. xlvii. 1864.

[‡] Velpeau (Méd Acad. des Sc., Dec. 1830) appears first to have hit upon the idea of curing aneurism by introducing needles into the sac; but I am not aware he ever actually carried his idea into practice.

College Hospital on this plan, no bleeding occurred, parietal suppuration was prevented by the use of Lister's antiseptic dressing, present relief was obtained, and the patient certainly continued alive a year after the operation.

(4) *Galvano-puncture*, originally suggested for external aneurisms, has in some instances proved thoroughly successful, even with vessels of considerable calibre,—as the external iliac.* But occasionally the results have been disastrous, and the application of the plan to the arch of the aorta would seem so imminently hazardous in various ways—among the rest through the well-nigh sure sequence of embolism—as to seem hardly justifiable. Still, in a case of prominent sacculation, promising external rupture *de die in diem*, an attempt to produce thrombosis on the side of the vessel disposed to give way, might, as an *ultima spes*, be made on this plan. Clotting (unless under exceptional conditions of the sac) is easily effected; while the support given to the yielding remnant of vessel-wall may ward off (as it appears to have actually done), imminent rupture. Looking at the matter *à priori*, the obstruction to further distension of the sac, in the direction specially brought under the influence of the thrombal process, would, however, be likely to give fresh activity to aneurismal enlargement in the adjoining parts, and, consequently, in the end add little to the total duration of life.

And so it has actually fallen out, that in the small number of cases, wherein the method has been actually tried, little real good has followed. It appears from a table copied, with additions, by John Duncan,† from the original document by Ciniselli,‡ that of four cases of aneurism of the thoracic aorta treated on this plan not one was cured. More recently M. Decristoforis has operated three times: the issue of the case is left in doubt in one of the three; in one of the others death took place by hæmorrhage in two days, in the remaining one, eleven days, after the operation.§ These deaths by hæmorrhage may, with probable fairness, be directly credited to the operation, on the principle a moment since suggested.

Yet more recently Ciniselli contributes five additional cases treated by electrolysis. In one only of the series was any real benefit secured; four of them prove that, when a large trunk

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[*] Eyre, Lancet, July, 1853.
[†] Ed. Med. Journal, April, 1866, p. 926.
[‡] On Electro-puncture of Aneurism, Cremona, 1856.
[§] Ed. Med. Journal, Dec., 1870, p. 537.

springs from the sac, the thrombal process is so interfered with, as to render the operation ineffectual, and of course to entail distant embolic impaction with all but certainty.* Ligature of the carotid and subclavian arteries should in such a case precede the galvano-puncture.

McCall Anderson records a very successful operation in a female who, five months after the fourth and last galvano-puncturation, "felt in the most perfect health," slight pulsation in the sac still remaining.†

On the whole, the operation may be said to have won its claim to consideration, where absolute rest and iodide of potassium have failed, and when rupture seems a matter of near probability.

For the operative details, the papers just referred to may be consulted; the pain endured has, in some instances, been fearfully severe. Richardson's spray was used with advantage by McC. Anderson.

(5) *Injection of the sac with astringent fluids.*—Since 1813, when the proceeding was first suggested by Monteggia, the injection of various fluids (tannic acid, alcohol, and acetate of lead by the inventor, acetic acid by Wardrop, perchloride of iron by Pravaz in 1853) into aneurismal sacs of vessels of the extremities, has from time to time been employed. The innominate artery was even made the subject of the bold experiment by Barrier. Successful in some instances, the general result of the operation has proved anything but encouraging: inflammation of the sac with peripheral abscess, rupture, hæmorrhage, and occasionally embolic gangrene of the limb, have destroyed life. The plan appears not to have been tried in the case of the thoracic aorta: nor could it be employed (even if combined with carotid and subclavian pressure), without imminent risk of the local and distant evils, we have just enumerated.

(6) *Pressure of the main vessels on the distal side of the sac.*—Experience has every now and then shown, that even spontaneous obliteration of *one* of the main cervical trunks communicating with an aneurismal arch, will not prevent the sac from steadily increasing in bulk. Yet relief to symptoms has occasionally followed artificial pressure (though only employed at intervals), to the carotid alone, in cases where the aorta has been proved *post mortem* to have been involved in the disease.‡

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* Medical Record, March 5, 1873.

† Ibid.; where also is a reference to a case electrolytically treated by H. I. Bowditch with present relief to suffering.

‡ *E. g.*, Lyon, Edin. Journal, Oct., 1847.

(7) *Ligature of a main vessel, or of both main vessels, on the distal side of the sac.*—The left carotid artery has been, at least three times, tied on the distal side of an aneurismal tumour, seated at the base of the neck, on the supposition that the sac in each instance sprang from a lower part of the ligatured trunk.[*] In all three instances relief of urgent symptoms and distinct prolongation of life were secured; though examination after death proved in every one of the number, that the carotid artery was unaffected, and that the sac really sprang from the arch of the aorta.

Relief of the more urgent pressure-symptoms of an aortic sac might, then, on the evidence of these cases, be fairly hoped for from ligature of the communicating cervical vessels. But much stronger grounds have been given for such expectation by the well-known case, in which C. Heath tied both the right common carotid and the right subclavian arteries of a woman, whose large sacculated aneurism rose from the right aspect of the ascending portion of the arch. The curative effects were most striking; and the patient, in spite of a mode of life the most dissolute and irregular, survived the operation for a little more than four years.[†]

(*b*) *Palliative.*—(1) The laryngeal symptoms of an undetected aortic aneurism have frequently been mistaken for evidences of disease in the windpipe itself, and tracheotomy vainly performed for their relief. T. W. Gairdner, however, endeavours to show, that the operation presents a fair claim to be admitted into the legitimate treatment of aortic aneurism,—"not unwillingly and as a last resource, but as early as it could be ascertained that laryngeal symptoms were the source of the more immediate danger." J. W. Begbie, in turn, advocates the proceeding, on the ground of its converting a very painful into a comparatively quiet death.[‡] I have had no personal experience of the operation in cases of aortic aneurism; but can conceive it might become justifiable as a means of *probably* prolonging life for a few days, where such prolongation might be a matter of importance.[§] And if the symptoms are, for the time being, *essentially* those of spasmodic laryngismus (*severe enough this may prove to place life in hourly jeopardy*), considerable relief might be temporarily obtained, and imminent death averted for a while: *under these special circumstances*, the operation was, indeed, twenty years ago recommended by Marshall Hall,[*] and would indubitably be not only a warrantable, but an advisable, proceeding.

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[*] By Tillanus and by Regin of Amsterdam, and by Montgomery of the Mauritius. Vide Cockle, Lancet, April 10, 1869, p. 489.

[†] Treatment of Intra-thoracic Aneurism, by the Distal Ligature. By C. Heath, 1871.

[‡] Ed. Med. Journal, 1858.

[§] A case recorded by Judd seems to show that life may by possibility be prolonged for several days, nearly a fortnight, by tracheotomy,—though the trachea suffers very serious pressure from a large sac: but obviously, we have no right to expect the repetition of such an extraordinary occurrence (Lancet, 1844).

(2) Arguing from the "extraordinary relief" to symptoms often afforded, when an enlarging sac, compressed by the clavicle, at length succeeds in partially dislocating the bone forwards, Stokes suggests that, where such displacement of the bone failed to take place spontaneously, division of the sterno-clavicular ligaments might be advantageously effected with the knife. I have certainly seen instances in which spontaneous luxation seemed to lessen the amount of suffering of these patients. In one instance, in particular, where the displacement of the sternal end of the bone was sufficient to put the attached portion of the sterno-mastoid muscle "on the stretch," the patient felt "getting better and better every day," and as though "nothing were the matter with him."[†]

801. In not a few cases of aortic aneurism the treatment becomes less that of the disease itself than of some associated or intercurrent state, as hypertrophy, or flabby weakness of the heart, or anæmia,—or of some sequential affection, such as obstinate bronchitis. Tartarised antimony, nitre, and digitalis in combination, are the best remedies for the latter malady.

III.—ANEURISM OF THE ABDOMINAL AORTA.

802. *Physical signs.*—(*a*) Inspection may, or may not, disclose some abnormal appearance. If the sac be small, and especially if it spring from the posterior aspect of the vessel, or if the aneurism be of the fusiform species and not bulky in any part of its extent, the eye may fail to detect any peculiarity in the form of the abdomen. If, on the other hand, the sac have acquired any size, pulsating prominence, of variable extent, is seen anteriorly, in the course mainly of the aorta, from the epigastrium downwards,[‡] or bearing especially to the left side of the abdomen, in

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[*] Lancet, Nov., 1852.

[†] Covey, U. C. H., Males, vol. xi., p. 316.

[‡] Hallington, U. C. H., Males, vol. viii., p. 61. The visible prominence here extended from one inch to the right of the umbilicus, to the anterior spine of the left ileum. After death dilatation of the aorta was found to commence an inch below the origin of the renal arteries; the sacculation implicated the lower part of the aorta and the common iliac.

rare instances to the right. The surface is smooth to the eye; the abdominal respiration-movements laterally, or bilaterally, impeded.

(*b*) The hand, placed on the anterior prominence, receives a single systolic impulse, sometimes of enormous force and quite out of proportion with the volume of the moving mass, while posteriorly, in the lumbar region, no trace of impulse may be perceptible. Generally speaking, if the abdominal walls be thin, the hands may be passed on either side of the sac, and an estimate formed of its bulk: the impulse is felt to be laterally, as well as anteriorly, expansile. The chief pulsation may be to the right of the spine, the sac sometimes mainly growing in that direction. The mass, fusiform, rounded, smooth or lobulated, is immoveable,[*] and commonly compressible more or less: caution is, however, requisite in ascertaining these particulars. In comparatively rare instances a second impulsive, or at least strongly jogging, action, diastolic in arterial time, may be felt: or, on the other hand, pulsation may be absent, when the sac is quite large enough to alter the form of the abdomen slightly; while, *per contra*, a very small sac sometimes furnishes powerful impulse. Thrill, systolic in the majority of cases, of diastolic time in rare instances, may be felt.

(*c*) The left semi-circumference of the abdomen may, or may not, be increased.

(*d*) The size of the sac can only, if at all, be accurately estimated by percussion; and the tenderness of the surface and the neighbouring parts generally often interferes with the process. Percussion of any force is dangerous and unjustifiable,—especially as intestinal note habitually interferes with precise limitation, vertically and horizontally [72]. Besides, a considerable time before death, the apparent size of the sac may be greatly increased by successive and repeated extravasations of blood behind the peritonæum.[†]

(*e*) I have heard in connection with aneurism thus seated: 1. A single systolic murmur, without sound of any other kind; 2. A dull muffled systolic sound, convertible into a murmur by a little pressure; 3. A sharp, abrupt, short systolic murmur at the left lumbar spine, much more marked than in front; 4. A systolic murmur below the sac, none immediately over it; 5. Occasionally a dull second sound. A murmur, diastolic in cardiac time, does not happen to appear in any of my notes as an observed fact; but inasmuch as murmur of this synchronism may occur in the vessel, when free from disease [751, *d*], there can be no question of its occasionally occurring, when it is aneurismal. 6. Double murmur may be well audible in front, and totally absent in the back.

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[*] In rare instances the mass is moveable; and in such cases, as Stokes points out, pulsation and even murmur may be made to appear and disappear.

[†] The extravasated blood may make its way to the front of the abdomen, and extend upwards to the pleura. Hallington, U. C. H., *loc. cit.* The extravasated coagula may pulsate distinctly under the influence of the sac, and with expansile character.—This passage is left exactly as it stood in the edition of 1854. Stokes (Op. cit., p. 627), in observing that I state that "the secondary tumours do not pulsate," has by some accident misinterpreted my words.

In some instances systolic murmur is audible in the reclining, when inaudible in the erect, posture: Corrigan supposes that in the earlier periods of the disease the hydrostatic pressure from above, by maintaining a state of tension of the sac, prevents the occurrence of murmur; while the diminution of pressure in decumbency, lessening the tension of the sac, allows of its production. Sometimes a moment's decumbency suffices to bring out the murmur well; at others the lapse of two or three minutes is required.* But I agree with Stokes, that this peculiarity will not help to establish the diagnosis between pre-aortic tumour and aneurism.† Not only is he right in supposing that the disappearance in the erect posture of an abdominal aortic murmur, which is audible in dorsal decumbency, will not distinguish aneurism from tumour, but I am certain, from recent observation, that even in simple abdominal pulsation the same effect may be produced by that change of attitude. On the other hand, it is indubitable, true aneurismal murmur sometimes disappears in the sitting and standing postures. The student must further remember that murmur may be totally absent in every possible posture.

The special characters of any existing murmur may vary as in the arch of the vessel. Its amount sometimes changes inversely as the growth of the aneurism: the noise may be so loud as to be audible at a little distance from the surface.‡

803. *Physical latency.*—It is all-important for the observer of an obscure abdominal disease to bear in mind, that even large-sized aneurism of the ventral aorta may exist *in the total absence of all positive physical signs*,—neither impulse, murmur, nor percussion-dulness being discoverable: the subjective symptoms are then very likely to deceive.

804. *Symptoms.*—(*a*) The patient may or may not be conscious

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* Dub. Journal Med. Science, vol. ii.

† Dis. of the Heart, p. 643.

‡ J. R. Reynolds, Med. Times, Sept., 1852, p. 284.

of pulsation, and this whether there be, or be not, objective evidence of morbid action of the kind. Subjective pulsation, absent for two years, in a case where it had long existed objectively, then suddenly came on after effort in running, and continued until death.

(*b*) Pain, following the course of nerves implicated by pressure, passing along the edge of the ileum down the thigh to the testicles and pudenda generally,—and in character raw, sore, pricking, cord-like, plunging, hot and burning at one time, cold at another, accompanied with spasmodic difficulty in passing urine, and with tonic contraction of the flexor muscles and inability to straighten the limb, the whole attended also with peculiar gnawing vertebral pain, existed in the case already more than once referred to. But obviously the neuralgic sufferings must vary with the exact site of the sac: sometimes they extend upwards to the left arm. In severity these pains present all varieties of degree: but the rule is they are violent, sometimes excruciatingly so. They vary in persistency, sometimes equably constant, oftener they increase paroxysmally,—and if so, especially by night. In some instances they disappear for a while completely: such relief may be traced to special changes of position, or it may be inexplicable. On the whole, pain is at once the symptom most distressing to the patient and most significant for the physician.

(*c*) Theoretically, anasarca of the lower limbs, or of one of them, must occur, according as the inferior cava or either iliac vein is pressed on; but in practice either effect is most rare,—and fulness of the subcutaneous abdominal veins from obstruction of the cava may exist without any pedal œdema even. Ascites of any clinically important amount is excessively rare, if indeed it ever occur through the influence of aneurism alone. The rarity of dropsy of any kind is in regard of diagnosis seriously significant.

(*d*) Pressure on the descending or transverse colon may obstruct the bowels, and cause flatulence, constipation and great labour in defecation. In the case of Hallington (loc. cit. p. 64) the sac was adherent to the descending colon by pseudo-cellular bands, and the calibre of the bowel in one spot much reduced by contraction of these bands.

(*e*) Wasting of the testicle has fallen under notice as a result of obliteration of the spermatic artery.

(*f*) The respiration, if the sac be of moderate size and seated low down, is of natural frequency and character; when high or of notable bulk, the tumour interferes with phrenic action, throws the onus on the upper ribs, and accelerates the act. Cough may be excited by mere pressure from below; but hæmoptysis does not occur, unless there be perforative communication with the lung.

(*g*) The pulse, even while the patient is visibly perishing from pain, insomnia and exhaustion, need not be accelerated,—ranging from 78 to 88: the pulse-respiration ratio may be a normal one of 4·35 : 1.

There does not seem to be any effective reason, why the radial pulses should differ from each other; yet, as will be seen by reference to the Plates in the Appendix, a specific difference may be steadily maintained between them,—when, of course, the absence of disease of the arch or of the innominate artery is to be assumed as proven.

The heart may be pushed upwards or sidewards.

(*h*) The temperature of the lower extremities may fall sensibly below that of the upper.

(*i*) The urine, sometimes ranging below the par of health in quantity, may be rendered albuminous by renal congestion induced by pressure on the emulgent vein; otherwise it is perfectly natural in composition as far as the aneurism is directly concerned; but, when the constitution begins to sympathize with the local mischief, I have observed continuous oxaluria.

(*k*) Seaton Reid has published a curious case, in which an aneurism arising opposite the cœliac axis, and separating the pleura from the diaphragm, seemed to be the only apparent condition (through traction of the great splanchnic nerve on the right side of the sac), explanatory of contraction of the right pupil. I have known dysphagia, apparently reflex, accompany abdominal aneurism seated in such manner as not to interfere mechanically with the diaphragm.

805. *Duration, and mode of death.*—In some instances these sacs have acquired enormous bulk: one preserved in the Fort Pitt Museum is said to have contained ten pounds' weight of coagula. Hence the inference that the gradual growth of the disease is not incompatible with existence. But we have little information as to the mean duration of life after the outbreak of symptoms: I have known twenty-four, thirty, and thirty-eight months intervene between the earliest indications and the fatal termination.

Death occurs by rupture of the sac into the peritonæum, or into the retro-peritonæal connective tissue, into the pleura, lung, colon, renal pelvis, or mediastinum; or, without rupture, by jaundice, gangrene, exhaustion from pain, insomnia, &c.

806. *Prognosis.*—People with aneurism of the abdominal aorta have in rare instances escaped that fatal termination, which is almost the necessary apanage of the disease. The sac has filled with stratified fibrine, and symptoms have wholly, or almost wholly, disappeared. Such patients have not then actually died of their aneurism, though unquestionably its existence may have rendered them more ready victims, than they otherwise would have proved, of the disease actually destroying them. And, further, complete repletion of a sac with coagulum, even to the level of the normal interior of the vessel, does not ensure removal of symptoms. All the evils, obstructive of function and excitant of pain, depending on abdominal tumour, may be felt as vividly as before; this has more than once been proved by *post-mortem* examination.

807. *Diagnosis.*—The diagnosis may be conveniently considered under the three heads of cases: First, where there are abdominal physical signs; secondly, where there are no physical signs, but serious ventral symptoms, mainly neurotic, with or without much constitutional sympathy; thirdly, where there are no signs, or very trifling ones, and no symptoms of the least apparent consequence.

I. *Abdominal physical signs existing.* — (*a*) The extreme difficulties sometimes arising in the distinction of mere *aortic pulsation* from aneurism have already been considered [755]. Curiously enough, the only case in which I have actually heard abdominal aortic murmur, diastolic in cardiac time, was one of diminished, instead of increased, calibre of the vessel.

(*b*) *Fæcal accumulation* is distinguished generally by the oval outline of the fulness; by its doughy inelastic feel; by the existence of several spots of toneless and toned resonance under percussion close to each other, and within the area of the swelling, from the intermixture of gas with solidified fæces; sometimes from the position of the mass; and, generally, from the history of the case. The pains of aneurism may be imperfectly imitated by those of peritoneal distension from the enlargement of the bowel; but it is rare indeed that a mass of fæces receives such arterial impulse from behind as to simulate that of aneurism.

(*c*) In the obscurity of their early symptoms, in the eventual pain, and in the gradual exhaustion they produce, there is considerable similarity between *lumbar and psoas abscesses* and aneurism; but the swelling of these abscesses passes in an elongated form from above downwards, and does not exhibit an irregularly globular shape, as aneurism sometimes does; they give neither impulse nor murmur. Tenderness exists in the lumbar spine, and there may be loss of motor power in the lower extremities; but the actual pain is materially less, as a rule, than in the aortic disease. Tubercle should be sought for in the lungs: the existence of phthisis would be directly in favour of lumbar abscess of tubercular origin,—against aneurism. It must not be forgotten that in rare instances lumbar abscess and abdominal aneurism have coexisted.

(*d*) *Hydronephrosis and pyelitic distension* are accompanied with renal symptoms, changes in the urine, tumour with the characters of renal enlargement,—a tumour of tuberous nodular outline, non-impulsive, murmurless, and extending further into the flank and into the back than aneurism. The urine may be albuminous in all three affections; but in aneurism it will not be purulent as in pyelitis.*

(*e*) *Tumours* of various kinds in the abdomen may pulsate in expansile manner, and be the seat of murmur; the murmur is high-pitched, whiffing invariably as far as I have heard; but a careful consideration of the whole case is the best safeguard against error. In a case of cancerous lumbar glands, seen some time since, not only were there expansile impulse and murmur, but such neuralgic pain as commonly accompanies aneurism; still the knowlege that a cancerous sarcocele had previously been removed prevented mistake. When there is an obvious solid mass, either a tumour or an aneurism, and which is pulsatile in the recumbent posture, the diagnosis may sometimes be made by placing the patient on the hands and knees; if aneurismal, the pulsation will remain; if that of tumour, conveyed from a healthy aorta, it will disappear, in consequence of the vessel and the morbid mass having ceased to be in close juxtaposition. But the presence of adhesion would greatly interfere with the successful employment of this test.

(*f*) *An enlarged lumbar vertebra*, pressing forward the aorta, will cause extra pulsation; but there is no lateral expansion of the vessel; and the murmur is whiffing or rasping, and not heard laterally.†

And, even if there be positive surety of the existence of abdominal aneurism, we are not at once entitled to pronounce it aortic; it may be situated in the cœliac axis, the hepatic, the superior mesenteric, or the renal arteries. But the signs of these aneurisms have as yet been imperfectly investigated,* and I know little of any one of them by experience. The distinction, even in regard of the application of compression in the treatment of the disease, is less important than might, on first thought, appear: for in truth the practical question will be, not so much in what vessel a given aneurism is seated, as whether the sac will, or will not, bear distal or proximal pressure.

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* Unfortunately for purposes of diagnosis a patient having enormous purulent accumulation in the distended pelvis and infundibula of one kidney may, in consequence of the communicating ureter being obliterated, pass perfectly normal urine,—that furnished by the fellow healthy kidney.

† Case by Dr. Taylor, in Author's work on Cancer, p. 528.

II. *Physical signs absent.*—Wherever obstinate abdominal neuralgic pains exist, especially in a male, and where the ordinary signs of visceral disease cannot be established, aneurism should be held in view as very probably present, even though there be no single physical sign to warrant such an opinion. Let the examination never be considered complete, however, without careful auscultation in the left vertebral groove. It will be necessary, too, to exclude, with as much certainty as possible, the presence of cancerous lumbar glands, and of cancer of the small intestines. An aneurismal patient of this class may be seen at a time when his general health is excellent, or already impaired.

III. *Physical signs and notable symptoms absent.*—Slight pain in the lumbar region may be the sole symptom for twelve months, to my own knowledge, after aneurismal distension has probably commenced. Hence the importance of thoroughly investigating physically cases of alleged "incurable lumbago and sciatica." Relief of lumbar pain by cupping will not disprove the dependence of that pain on aneurism.

Whether any diagnostic help may be obtained from the sphygmograph, remains to be determined [*vide* APPENDIX].

808. *Treatment: (a) Medical.*—The medical treatment is the same as of intra-thoracic aneurisms in general. Iodide of potassium especially would claim careful trial.

Rest, as complete as can be managed without serious disturbance of the digestive organs, is, beyond doubt, of extreme importance in cases of the class. There are many instances on record in periodical journals, where perfect quiet so singularly improved the local and general state, as to make the patient believe himself free from disease, and consequently to commit acts of imprudence which brought back the previous symptoms with increased intensity. Were the patient's endurance equal to the occasion, and were he made to fully comprehend the haven of cure within his possible reach, actual recovery, in a practical sense, might be occasionally secured by absolute and continued rest.

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* Ballard, Physical Diagnosis of Dis. of the Abdomen, p. 217.

809. (*b*) *Surgical.* *a. Pressure on the proximal side.*—The remarkable case, in which W. Murray[*] first submitted the abdominal aorta to tourniquet-pressure on the cardiac side of a large aneurismal sac, marks an epoch in the management of the disease. Though suggested previously, the plan does not appear to have been put to the practical test before. The patient said he "felt as well as ever he did in his life" five months after the operation: he, however, "complained of slight weakness and pain in his back and legs," but was able to work hard as an engineer. He eventually died six years later, from rupture of another aneurism above the occluded spot; whether the additional disease did, or did not, exist at the time of the operation, there is no evidence to show. While the pressure was applied, the patient was kept under the influence of chloroform; by this means rapid and lumpy, instead of slow and laminated, thrombosis was secured.[†]

Another successful case has recently been placed on record by W. Moxon and A. E. Durham.[‡] In this case most probably, in Murray's certainly, the sac occupied the lower part of the aorta, involving the origin of the inferior mesenteric.

β. Pressure on the distal side.—A plasterer, aged twenty-seven, suffering from abdominal aneurism, involving the cœliac axis and origin of the superior mesenteric artery, was submitted by Pavy and Bryant,[§] to pressure by Lister's tourniquet, applied for twelve hours, and again for four hours after twelve hours' intermission, to the vessel at its bifurcation. The patient died of peritonitis, evidently caused by pressure-contusion, eleven hours after the removal of the tourniquet. Very effective thrombosis had been produced within the sac.

GROUP D.—DISSECTING ANEURISMS.

810. Dissecting aneurisms (*vide* Diagram, p. 476, fig. 9) differ variously and completely from the other species of the disease.

811. The anatomical conditions of abrupt tearing of the coats of the aorta, with filtration of blood between them, are, in their

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[*] Med. Chir. Trans., vol. xlvii. 1864.

[†] Rapid Cure of Aneurism by Pressure, by W. Murray, 1871.

[‡] Med. Chir. Trans. vol. lv. 1872.

[§] Med. Chir. Trans., vol. iv. 1872.

principal varieties, demonstrable with fair precision from existing records. But the clinical history has as yet been very imperfectly worked out. And, indeed, from the nature of things, it seems singularly unlikely that any general account, applicable even to the majority of such cases, can be given,—seeing that the symptoms must in great part depend upon the extent and precise portion of the aorta affected.

812. *Symptoms.*—The symptoms in published cases may clearly be referred to three heads, which the observer should always aim at severally distinguishing :—namely, (*a*) symptoms of shock to the system at large; (*b*) of dynamic and statical disturbance of the injured artery; and (*c*) of mechanical interference with the function of organs supplied by branches from the part of the vessel involved.

(*a*) The symptoms of shock are, primarily, sudden faintness or actual syncope, and, on recovery of consciousness, nausea, vomiting, and pain in the thorax or abdomen; secondarily, febrile action, by no means necessarily very marked, thirst, furred tongue, and abdominal tympanitis.

(*b*) The dynamic disturbances of the artery are signified by more or less severe pain in its course, and throbbing action, irregular in force and rhythm. Statically, the vessel and its injured branches are widened and rendered uneven; while the obstruction to the current, offered by the prominent and ragged lining membrane in the site of its ruptures, gives rise to blowing systolic murmur, which, if seated near the heart, may be mistaken for that of constrictive disease of the aortic orifice.

(*c*) The group of mechanical origin are produced by the accumulation of the blood, filtrated between the coats of the aorta, against the orifices of arterial branches, whereby these are completely, or almost completely, blocked up. The nature of these symptoms will, of course, depend on the distribution of the blocked-up vessels. Thus, in a remarkable case, observed by R. B. Todd (Med. Chir. Trans., vol. xxvii.), where the innominate and the renal arteries were mainly obstructed, very singular cerebral symptoms and suppression of urine marked the event. If a main bronchus were pressed on by the suddenly enlarged vessel, equally sudden unilateral deficiency of breathing (the percussion-sound remaining at first unaltered), would ensue.*

813. In not a few recorded cases there were no conditions of the heart or great vessels, known to exist prior to the actual

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* Vide also a case of Risdon Bennett, Med. Chir. Trans., vol. xxxii.

event, which might have led the observer even to suspect dissection of the arterial coats as a likely or even intelligible occurrence. In certain rarer cases the existence of such previous diseases has been known, and such knowledge would materially aid in the diagnosis. This is especially true of special morbid conditions,—as, for instance, where the arch of the aorta was known to be the subject of serious coarctation [826].

Two cases of injury to the aorta have fallen under my notice, as the results of railway concussion, in which all reasonable proof was obtained, except the final and conclusive evidence of *post-mortem* examination, that dissection of the aortic coats on a limited scale had been abruptly effected.

814. *Treatment.*—Were the practitioner fortunate enough (guided by the sudden supervention of symptoms of the three classes just distinguished, and of a strong arterial murmur in a person known to have previously been free from this physical sign), to divine the occurrence of acute separation of the coats of the aorta, it does not appear that, in the present state of knowlege, the management of the case would be materially improved by his sagacity. Did he fail to diagnose the occurrence, his aim would be to restore the patient from the first shock of the accident, control excited arterial action, and relieve symptoms as they arose. And it does not appear that art could do more than this, were the anatomical nature of the affection understood from the first. True, the free administration of ammonia, as a solvent for thrombal deposits on the floating edges of the torn inner coat, might be theoretically insisted on, were the injury to the vessel surmised, or, *a fortiori*, diagnosed: but ammonia would in infinite probability be prescribed, in total ignorance of the damaged state of the aorta, from faith in its efficacy as a diffusible stimulant.

IV.—VARICOSE ANEURISMS OF THE ARCH OF THE AORTA.

A.—COMMUNICATION WITH THE SUPERIOR VENA CAVA.

815. A woman, aged fifty, having suffered for some years from aneurismal symptoms, one evening, while stooping at laborious work, suddenly felt as if strangled, changed colour, felt giddy, and sat up all night in dread of suffocation. When seen the next
ce and upper part of the body were deeply cyanosed,
ies of the upper cava enlarged, those of the lower
e ordinary signs of aneurism of the arch existed about

the right infra-clavicular region. Mayne, the observer of the case, arguing from the existence of a powerful superficial whirring systolic murmur [243], loudest at the second right cartilage, accompanied with thrill, perceptible not only at the spot, but, in spite of great œdema and swelling, over the right internal jugular and subclavian veins, and taking into consideration the extreme suddenness with which the serious symptoms above referred to set in, made the diagnosis of an aortic aneurism communicating with the superior cava. The pulse full, strong, and jerking, beat about 110 in the minute.

Death ensuing on the tenth day from the patient's sudden seizure, an opening "in size and shape resembling the button hole of a shirt, and crossed about the centre by a delicate frenum," was found between the vena cava and an enormous globular dilatation of the arch.* The heart exhibited nothing abnormal: the jerking pulse, it may therefore be presumed, depended directly on the loss of arterial current, sustained through escape of blood from the sac into the vein; but such mechanism is so singularly rare that the occurrence of jerking and visible pulse in connection with it can scarcely be held to invalidate the significance of the sign in relation to aortic reflux [631].

B.—COMMUNICATION WITH THE PULMONARY ARTERY.

816. If an individual, known, or not known, to have been the subject of aortic aneurism, suddenly experience after effort a sensation of something giving way in the cardiac region, feel faint, become pale and exhibit the general characters of nervous shock to the heart, followed by peculiar fluttering in the chest,—if he subsequently suffer from dyspnœa to orthopnœa, more or less cyanotic blueness of the lips, pallor of the face, chilliness, prostration of strength, anxiety, terrible dreams, occasional nausea and vomiting, syncopal and pseudo-epileptic attacks, and become anasarcous in the lower extremities, while the lungs and liver undergo mechanical engorgement, as proved by percussion,—if all this coexist with powerful systolic thrill, limited to the second and third left interspaces close to the sternum,† and with loud whirring murmur essentially systolic and intermittent, though sometimes inclining to continuousness (or, it may be, double

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* Dublin Hospital Gazette, Feb. 1854.

† This limited site will be lower in position, somewhat, if the heart itself be lowered by hypertrophy.

murmur similarly seated), the diagnosis of sudden communication between the aorta and either the pulmonary artery or right ventricle is warranted. The further determination of the existence or not of aortic aneurismal dilatation must turn on its own special signs.

817. F. W. Wade has published a case, from which he appears to infer that non-conduction of existent diastolic murmur downwards to the heart's apex is the key to the diagnosis of aortic communication with the pulmonary artery.* But this inference seems to me very seriously shaken by the fact that in some cases of aortic regurgitation the attendant diastolic murmur is not transmitted to the left apex,—where, on the contrary, a pure second sound is heard: this holds true even of excessively loud basic reflux murmur [115]. Yet more, in a case of aorto-pulmonary communication by W. Roberts,† the attendant murmur (a double one) was distinctly heard at the left apex.

818. In W. Roberts' case, wherein the communication had evidently been effected, not by a sudden rent, but by gradual thinning down, of the aneurismal wall, the maximum point of the murmur corresponded exactly to the left limit of the pulmonary artery, and diminished markedly in all directions from that spot. Double, loud, rasping, superficial, audible at an inch from the surface, the murmur was attended with intense tactile thrill. The pulse, markedly jerking, was visible in all the superficial arteries.

The observer of this case supplies a sphygmogram of the radial pulse, remarkable for the great height and perfect verticalness of the upstroke, the downstroke being highly dicrotous. The tracing is wholly wanting in the flattened summit usually found in connection with rigid vessels and aneurismal dilatation near the heart, and, apparently, according to the narrator, indicating aortic reflux alone, is *pro tanto* deceptive.

819. *Mode of origin.*—Habitually sudden, actually instantaneous, in its occurrence, the communication may, on the contrary, be slowly effected by a gradually erosive process. If the communication arise in this gradual fashion, the attendant symptoms may be wanting in the elements of sudden cardiac shock and respiratory distress, which are so striking, when abrupt rent of the vessels concerned throws their two channels into one.

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* Med. Chir. Trans., vol. xlv.

† Brit. Med. Journal, May, 1868.

820. *Duration of life.*—Recorded cases[*] are, as yet, too few in number to supply trustworthy information concerning the possible duration of life after the communication has been accomplished. A history, printed by R. W. Smith, seems to show that existence may be prolonged for three months; here the patient died in a pseudo-epileptic fit.[†] I have before me a preparation in which the vessels communicate by an opening, round, smooth, and half an inch in diameter on the aortic side: round, of the size of a split pea on the pulmonary side:[‡] according to the catalogue, the existence of the aperture was "not detected during life by any morbid sound or symptom." And it is remarkable enough that in Wade's case the symptoms, at the moment the communication between the two vessels was probably effected, were but slight: and the patient left the hospital after "a two or three weeks'" sojourn, "declaring himself well." He eventually perished about a week afterwards from a fresh rent, extending from the sac into the right ventricle; the communication with the pulmonary artery bore the impress of non-recency. In Roberts' case the special signs of the affection certainly existed eight weeks before death. On the other hand, in three recorded instances death took place in four minutes, in nine hours, and in twelve hours respectively, after the presumed time of rupture.

821. *Mode of death.*—In protracted cases death has occurred from exhaustion, through insomnia, restlessness, dyspnœa, pulmonary congestion, and general dropsy.

C.—COMMUNICATION WITH THE RIGHT VENTRICLE.

822. A man, aged twenty-five, in his ordinary state of health, felt a "crack in the heart," and became faint and pale, while lifting a sack of flour. Though very ill, he continued at work for three or four days. Nine weeks later, Hope found the face bloated and purplish, the legs very, the hands slightly, œdematous, the pulse 80, and excessively jerking; no pain; the least effort caused dyspnœa and irregularity of pulse for two or three minutes. The following physical signs also existed,—thrill in the third interspace,[§] two inches from the sternum, with very loud superficial sawing murmur, like a whispered *r* at the same spot, most

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[*] Vide an additional one by Hughes Bennett (Clinical Medicine).

[†] Dublin Journal, vol. xviii.

[‡] U. C. Museum, No. 2254.

[§] Although this is not stated in Hope's narrative, the left interspace is evidently meant;—Diseases of the Heart.

marked in systole, less in diastole, with also a continuous rumbling sound,—no thrill nor murmur above the clavicles, impulse of the heart not materially increased. The patient died, highly anasarcous, nine weeks after the examination. A sac, as large as a small hen's egg, immediately above the aortic valves, opened by two small apertures into the right ventricle.

In a male, aged thirty-one, observed by T. S. Beck, both sounds are represented to have been distinctly audible; and immediately following, and loudest just after, the second sound, a sawing murmur disagreeably close to the ear, accompanied with thrill, most distinct at the base of the heart near to the sternum,—but at which side of that bone is not stated,—was heard.* Here, in addition to an opening between the aorta and right ventricle, there was a communication between both ventricles.

D.—COMMUNICATION WITH THE RIGHT AURICLE.

823. There is reason to believe that where communication is established between an aneurism of the arch and the right auricle, the attendant thrill and murmur will be of maximum strength in the second and third right interspaces.

824. Little, if anything, is known of the duration of life subsequent to this perforation. An ostler, aged thirty, was brought in dead to University College Hospital at about half-past ten p.m.; that evening he had been heard to groan by a fellow-workman, and answered to inquiries as to the cause, that "he should soon be better;" after this, having taken some tea and a small quantity of gin, he fell in the street, while returning to work at about ten p.m. and died almost immediately. An aneurism of the ascending aorta, three inches in diameter, had opened by a rupture, jagged on its aneurismal, smooth on its auricular surface, into the right auricle,—when opened out, the communication seemed about as large as a fourpenny piece.†

The man had been known to have suffered for a long time from pain in the chest, increased by stooping; still he was looked upon by those who knew him as a healthy person: the aneurism alone, it seems evident, did not seriously disturb him. The rupture was, in all probability, partially effected at the time of the groan, completed at the moment of the fall.

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* Med. Chir. Trans., vol. xxv.

† U. C. Museum, No. 4026, described by St. John Edwards, "Medical Times," Dec. 1851.

E.—COMMUNICATION WITH THE LEFT CARDIAC CAVITIES.

825. I have not met with any example of communication by rupture between an aneurism of the aorta and the ventricle or auricle of its own side of the heart. Either occurrence, especially the former, is indeed difficult enough of conception.

§ VI.—COARCTATION AND OBLITERATION OF THE AORTA.

A.—ARCH OF THE AORTA.

826. The arch of the aorta is subject to a process of closure, more or less complete, either as a result of imperfection in the developmental changes occurring more or less immediately after birth (*paulo-post congenital variety*), or as an effect of disease in adult life (*acquired variety*).

I.—PAULO-POST CONGENITAL VARIETY.

827. The closure of the ductus arteriosus, commencing after a few respirations, is, according to Bernt, completed in some cases by the third or fourth day, by the eighth in half the number, and by the tenth in all. But the statements made by various other observers refuse to be reconciled with the deductions of Bernt; the latest researches on the question, especially those of Alvarenga, lead to a very different view of the rapidity, with which closure is effected. The Lisbon professor found that in 54 cases examined between the moment of birth and the twenty-eighth day, no single example of perfected occlusion existed; of 19 cases, where death occurred between the twenth-ninth and thirtieth days, one only exhibited occlusion; and among the bodies of 40 subjects, dying between the forty-fifth day and the twelfth year, the duct was closed in 15, still pervious in 25.[*]

828. Whatever be the rate at which the process of closure advances (and it is evidently subject to great variations), the constrictive action, in the *normal* course of events exercises no influence on the contiguous portion of the arch of the aorta; but in a certain number of instances the contracting process *abnormally* extends thereto.[†] Whatever is the mechanism of the coarctation,

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[*] Quoted in Dobell's Reports, vol. ii., p. 126. 1870.

[†] Objections have, however, been taken to this idea of the mechanism of the constriction by Chevers,—also by Rokitansky, who adopts Reynaud's theory of dependence on perverted development of the branchial arches where continuous with the aorta.

the effective agency probably comes into play very shortly after birth, and is in time *paulo-post congenital.*

829. The constriction, commonly seated at, or very close to, the ductus arteriosus, varies in length, but unless in very exceptional instances averages scarcely more than a line or two, as though a string had been tied round the part,—varies also in amount from more or less considerable narrowing to complete obliteration. In the latter case the vessel presents the appearance of a ligamentous cord; in the former the internal surface is, except in the rarest instances, smooth. Vegetations, when present, must greatly intensify murmur.

830. The vessels springing from the arch are always more or less, sometimes enormously, dilated; and various second and third-class arteries, especially the deep cervical, intercostal, and epigastric similarly widened. Local dilatations, in addition to the general enlargement, simulate so many aneurismal sacs,—as has particularly been seen in the intercostal arteries.*

831. Congenital deficiency of the aortic valves, and irregular distribution of the ulnar, and certain other, arteries have been witnessed.

832. *Clinical Course.*—In respect of clinical course, past experience divides these cases into four classes:—

A class in which no special symptoms attracted attention during life, and the constriction has been a *post-mortem* discovery.

A second, in which symptoms of disease existed, or were supposed to exist.

A third, wherein disturbances of the circulation were more or less confidently referred to an imaginary aneurism of the arch.

And a fourth, in which the physical signs, conjoined with slight symptoms, led to the true diagnosis.

833. *Symptoms.*—Separating what is wholly contingent and accidental from the real functional effects of constriction, the following symptoms may be admitted, as really appertaining to the narrow state of the vessel.

(1) Fits of dyspnœa, dry irritable cough, and occasional hæmoptysis occur,—but only when the heart itself is unusually excited, or under the influence of more or less violent strain on the circulation. Uneasy sensations may be experienced within the chest, probably both in the aorta itself [810] and in the cardiac plexus, —but actual pain is, at the least, rare.

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* Vide fig. by Alex. Meckel, copied into Craigie's Memoir, Ed. Med. and Surg. Journal, Oct. 1841; also the opposite diagram.

(2) As effects of impeded circulation in the brain, fulness in the head, cephalalgia, vertigo, and epistaxis occasionally present themselves.

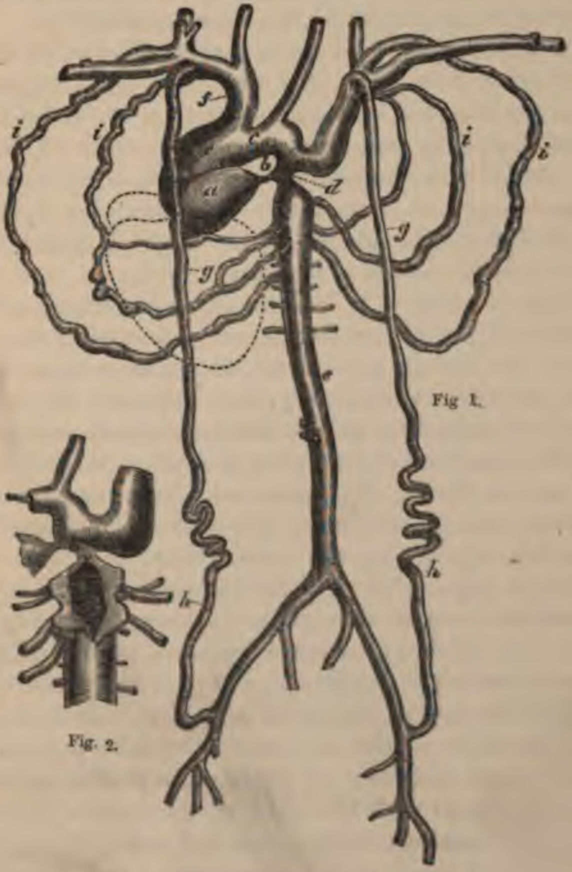

Fig 1.

Fig. 2.

(*After an unpublished drawing by Carswell.*)

Fig. 1, *a*, pulmonary artery; *b*, arterial duct; *c*, arch of aorta; *d*, coarctation of aorta; *e*, descending aorta; *f*, innominate artery; *g*, internal mammary artery; *h*, epigastric artery; *i*, *i*, *i*, *i*, deep-seated arteries of neck and intercostals, forming, together with the internal mammary and epigastric arteries, a collateral circulation with the thoracic and abdominal aorta and internal iliacs.

Fig. 2. Aorta laid open, showing by probe the amount of constriction.

(3) There seems no disposition to stagnation within the systemic capillaries (just as in aneurism of the arch also); even œdema of the feet fails to occur, unless as a sequence of intermediate obstruction in the lungs and right heart. Neither does the urine become albuminous.

(4) The influence exercised on general nutrition seems less than might be anticipated; in the case, which I described some years ago, the four extremities, especially the lower, were very fairly nourished,—a fact the more remarkable in regard of the latter as they lived wholly, or almost wholly, an *anastomotic life.*

(5) It seems scarcely probable a constricted state of the aorta shall generate a new, or modify any existing, diathetic state: tubercle in the lung has more than once been noticed,—but, it is fairly presumeable, by accidental association only. Low inflammations, especially in the inferior extremities, might be theoretically looked for; but have not to my knowledge been found. The man, who fell under my notice, had slight lymphatic inflammation and whitlow in one of his upper limbs.

834. *Physical signs.*—(1) Not the least important and readily ascertainable of the physical signs, are presented by the extreme pulsation of the carotid, sub-clavian, temporal, deep cervical, and intercostal arteries. Occasionally local expansile impulse, aneurismal to the feel (and sometimes strong enough to gradually wear away the ribs), may be felt from place to place in the latter vessels. Further, even at the top of the sternum, if the innominate artery and the aorta itself on the cardiac side of the constriction be much dilated, undue impulse may reach the surface.

(2) Murmur may be heard of maximum intensity at the second right interspace close to the sternum; harsh; of sawing special character; high-pitched; conveyed upwards, to the right and to the left,—downwards in the direction of both ventricles, but more to the right than the left,—backwards to both vertebral grooves, but more distinctly to the right than the left; persistent; not notably influenced by change of posture; in rhythm synchronous at its commencement with the close of the heart's first sound; continuing after this sound has ceased, and not covering it during the brief moment while both are coexistent; and, when listened to at the base of the heart, striking the ear as though it originated at a point more or less distant from the site of production of the heart's systolic sound. Such were the characters of the murmur in the case to be by-and-by more fully spoken of. Probably generated by the passage of blood through the coarctation, this murmur might possibly also depend on rippling of the current in the widened part of the arch. In this case the heart's own sounds were well audible.

(3) There is no visible prominence at the top of the sternum.

(4) Thrill may be felt at the second right, less at the second

left, interspace,—sometimes above the clavicles, and also in the site of the pulsating prominences of the intercostal vessels. Jugular pulsation of cardio-respiratory rhythm may be seen.

(5) Percussion at the top of the chest either gives normal results; or, if the innominata and the arch on the cardiac side of the obstruction be much dilated, some slight unnatural dulness may be detected.

835. *Causes.*—Of the causes indirectly leading to the coarctation nothing is known. It has hitherto been seen with greater frequency in males than females, in a ratio of about 3 : 1.

836. *Issue and prognosis.*—That aortic coarctation is not necessarily a malformation of fatal tendency, appears sufficiently from the fact that it has in eight or ten instances been found in the bodies of persons dying at upwards of fifty years of age, and in the case recorded by Reynaud, the individual had actually attained the patriarchal term of ninety-two.

Yet, on the other hand, the prognosis is always uncertain; the annals of cases show, that death may take place in the midst of robust health, with scarcely a moment's warning. The patient may perish in one of four main ways. First, he may be cut off suddenly (through the aortic affection) by rupture of the vessel itself, by rupture of the heart, or by formation and rupture of dissecting aortic aneurism.* One recorded case, at least (that of Meckel), shows that rupture may occur, where habitual quietude of impulse seemed to render such a catastrophe a most unlikely event. Secondly, he may die of cerebral disturbance or pulmonary congestion, pneumonia or bronchitis, or of dilated heart,—dependencies on the aortic obstruction. Thirdly, he may perish through acute endocarditis and aortitis, with embolic and thrombal obstruction of various subsidiary vessels.† And, fourthly, he may be cut off by some completely unconnected, cardiac or other, disease.

837. *Diagnosis.*—If a systolic, or rather slightly post-systolic murmur, following the track of the aorta and of maximum force above the heart's base, were discovered as a permanent condition in a person free from spanæmia and from any of the ordinary murmur-producing affections of the valves, and if there were no local bulging, and at the most but slight abnormal dulness at the

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* Barker, Med. Chir. Trans., vol. lxiii., p. 131.

† As was the fact in a very interesting case, the main particulars of which were kindly forwarded me (June, 1867) by Dr. Campbell, of Montreal. Eleven years before death the coarctation had been diagnosed in this case by Camman of New York.

top of the sternum, while the patient suffered neither from the local symptoms nor the concentric pressure-signs of aneurism or mediastinal tumour, nor from cyanosis, there would be fair motive for strongly suspecting the existence of coarctation of the arch. This suspicion would be converted into certainty, if throbbing of dilated and sacculated cervical and intercostal arteries were more or less markedly present. But were this latter condition absent, the surety of the diagnosis would fail, inasmuch as all the signs previously rehearsed might depend on an obstruction at the aortic orifice of the innominate artery.

The diagnosis of the affection has so rarely been even attempted, that it may not be without advantage to reproduce, here, the main facts and arguments which appeared to establish it in a case, that came under my notice some years ago.[*]

> "Such are the characters of the murmur heard in Gunnee [vide 834 (2)]. What are the conditions of the heart's sounds? Both are audible at the right and the left apex and at the base; while, of the two, the second possesses in the more satisfactory manner the attributes of health. Now the perfect freedom of the diastolic sound from morbid character proves the absence of mitral constriction, of tricuspid constriction, and of regurgitation through the aortic or pulmonary orifices. On the other hand, of the conditions, possibly productive of *quasi*-systolic (really slightly *post*-systolic) murmur, mitral and tricuspid regurgitation require only to be named to be rejected as the essential causes of that audible in Gunnee's chest. Compare the characters of the murmur, a moment since rehearsed, with those of an acknowledged mitral or tricuspid regurgitant murmur, and you will, if on first thought disposed to question the propriety of this absolute rejection, at once admit its justness. But what is to be said of pulmonary or aortic constriction? Is the murmur, we hear, the result of obstruction of one or other, or of both the arterial orifices? The synchronism, localization, and other characters of the murmur, loosely considered, seem not discordant with this explanation of its mechanism. But, examined at all closely, the hypothesis proves untenable. The murmur cannot be pulmonary constrictive, in truth, because (*a*) it is not perfectly synchronous with the first sound; because (*b*) it is too loudly transmitted down the chest; because (*c*) the maximum point lies to the right of the sternum; because (*d*) the first sound is too well characterised, too distinctly audible; and, besides, the pulmonary orifice can scarcely be constricted, because (*e*) cyanotic symptoms are totally wanting. Nor do I think the murmur indicative of aortic obstruction. Three of the objections just urged apply here also—those marked *a*, *b*, and *d*. Besides an aortic murmur is of maximum force at the heart's base,—not, as that under examination, at the second space to the right of the sternum. And, again, the pitch of the murmur indicates, as I think, with much certainty, a *considerable* amount of constriction—that the spot where it is produced is narrow. Now we cannot admit that such constriction exists at the mouth of the aorta, for the radial pulses are at the least fairly broad and full. If the aorta be narrowed, the lessened calibre must affect, not the orifice, but some spot in its course, so placed as to exercise no influence on the radial arteries. Should the validity of this argument be demurred to, on the score that the large vessels at the base of the neck, springing from the arch, are (as is indubitably the fact) materially dilated, and that through this dilatation the influence of co-arctation of the sigmoid orifice of the aorta must be annulled, that counter-argument would avail its employer nothing. For, inasmuch as that counter-argument assumes as its basis the co-existence of two conditions (*narrowed* aortic mouth with *widened* arch and connected great vessels) which Nature, so far as I can ascertain, has never been known to unite, it falls at once to the ground, and cannot, indeed, be said to have any logical existence at all.

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[*] Med. Times and Gazette, Oct. 17, 1857.

"It would seem, then, that the ordinary causes of cardiac murmur fail to explain that heard in Gunnee. Let us next look *seriatim* to the rarer, the more extraordinary mechanisms, coupling with our consideration of these that of the class of arterial causes.

"Can the murmur be caused by the flow of blood through a *patent foramen ovale?* That such flow is, independently of other conditions, capable of generating murmur may reasonably be, and commonly is, doubted. *Rationally* the notion may be opposed ; for an intra-auricular current must be wanting in force, and the edges of the opening, through which the blood flows, are smooth. *Experience*, on the other hand, may point to two series of cases of this class : in the one murmur existed, in the other it was absent. Now, in the first of these series other abnormal states, especially constriction of the orifice of the pulmonary artery, were present to plausibly explain the murmur. In the second the absence of murmur would seem to demonstrate the unfitness of mere patency of the foramen ovale to produce abnormal sound : but only seems ; for it must be confessed the evidence (instance Bizot's cases) is not so convincing as might be desired ; there is habitually a want of proof, that when the foramen has been found widely open after death, with an alleged deficiency of physical signs during life, clinical examinations had been made with sufficient care to justify the unqualified assertion of that deficiency. And lately, indeed, W. O. Markham has published a case in which a well-marked murmur, *apparently* inexplicable in any other way, seems to its observer to have been generated at an open foramen ; but conceding, *argumentia gratiâ*, that this murmur was really so generated, we cannot utilise the case in accounting for that heard in Gunnee—inasmuch as in our patient the murmur attains its maximum intensity to the right—in Markham's was loudest to the left—of the sternum. [691, *f.*]

"*True sacculated aneurism of the left ventricle* has in rare instances been attended with murmur : but that murmur has been of maximum force about the left apex, and has synchronised precisely with the systole, simulating the essential sign of mitral regurgitation. Have we not here ground enough, without any reference to the adjuncts of Gunnee's murmur, to show that this cannot depend on an aneurismal ventricle ?

"*Calcification of the arch* may give rise to murmur, systolic or slightly post-systolic in time : such murmur might of course reach its maximum, as here, at the second right space : while jerking and forcible impulse at the sternal notch, and thrill, somewhere in the course of the arch, as observed in Gunnee, are among its pretty frequent accompaniments. But the intensity of Gunnee's murmur, its extremely elevated pitch, and its notable transmission downwards are, according to my experience, discordant with the idea of this mode of generation.

"Nor can I conceive the murmur under examination to be produced by the pressure of a *mediastinal tumour* against the arch. It is singular enough, I may observe parenthetically, how rarely intra-aortic murmur is thus generated in that portion of the vessel. Still it sometimes is so generated. Why not here ? Because the pitch is too high, the intensity too great, the downward transmission too marked, of the murmur we are called upon to explain. Besides, were tumour-pressure the cause of the murmur, other pressure-signs, concentric, eccentric, or both (not one of which is here discoverable), would assuredly be present.

"More serious seem the claims of that most uncommon state, *coarctation* on the cardiac side *of the orifice of the innominata.* For, observe, the pitch, the intensity, the synchronism of the murmur are perfectly accordant, the site of maximum development not discordant, with mechanism of the sort ; while the apparently disconnected localization of the first sound and the murmur at the base of the heart also deposes in its favour. And, again, the thrill at the second right space, coupled with the negation of intra-thoracic pressure-signs, all tell in the same direction. Still we have not hit on the true cause yet. For, in the first place, a murmur of this origin would not be conveyed downwards, and would be loudly audible in the right carotid ; and in the second place (if you will forgive my torturing the words of Pope),* I think I may say, forcible though the arguments for narrowed innominata in Gunnée prove, 'Look in *his neck*, and you forget them all.' In truth, were the innominata constricted, what should the condition of cervical pulsation be? Why, obviously, there should be nothing abnormal in the impulse of the left carotid and subclavian arteries; while the expansion and beat of those vessels on the right side should be more or less materially below par. Now, here, on the contrary, the impulse and expansion are largely in excess on both sides of the neck; and that excess is somewhat greater on the right than on the left.

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* "If to her share some female errors fall,
Look in her face, and you forget them all."
Rape of the Lock.

"The site and peculiar rhythm of the murmur might be indicative of an *aneurism* of one of the *sinuses of Valsalva;* but its intensity, high pitch, and downward transmission seem irreconcileable with this view of its mechanism. Again, the site, the rhythm, and the intensity of the murmur, coupled with the percussion-dulness at the top of the chest, and the sense of deep-seated impulse in the same situation, bear witness in favour of ordinary *dilated or sacculated aneurism of the arch*; while, *per contra*, the pitch of the murmur, and the remarkable identity of its characters, during the whole time the patient has been under observation, taken in conjunction with the deficiency of pressure-signs, and the absence of specific pain either in the vertebral region or in the front of the chest, militate forcibly against the real existence of that disease. The unchangeableness of the attributes of the murmur through so long a period I imagine to be peculiarly hostile to the notion of aneurismal mechanism. Still, it must be confessed, the sum of objections fail to show the impossibility of the presence of aneurism; and, as we shall presently see, the truth is, that though the diseased state is not essentially aneurismal, aneurism of a certain fashion has actually arisen, and still exists, as one of the consequences of that state. *Dissecting aneurism* of the arch need only be named to be rejected. True, for ought I know, a murmur, such as that heard here, might appertain to that form of mischief in the vessel; but there is a total want of signs of obstruction of the blood-current through the vessels connected with the arch,—and to settle the matter at once, we have no experience of dissecting aneurism as a disease of chronic course.

"For the existence of some one of the varieties of *varicose aneurism of the arch* we have this *primâ-facie* argument, that such aneurisms are generally small; in other words, the absence of notable dulness, of pressure-signs, and of specific pain in Gunnee do not throw the difficulty in the way of admitting the presence of a varicose, that they do of a simple, aneurism. Well, first, can we admit here the existence of a small aortic sac communicating with the *cava superior?* No. For the limited experience hitherto obtained of this compound affection entitles us to look *inter alia* for a whirring quality in the attendant murmur, for cyanosis, for grave symptoms, dyspnœa, œdema of the neck and upper chest, distension of the jugulars, and various other indications of an amount of venous obstruction menacing existence. It is, indeed, doubtful whether life could be prolonged for any length of time. Secondly, did a communication exist with the *pulmonary artery*, the murmur would be precisely systolic; I doubt its being by possibility so high-pitched as in the case of Gunnee; it would be attended with more marked thrill; and assuredly we should expect subjective and objective symptoms of graver character than those we have had to set forth. Thirdly and fourthly, communications of an aortic sac with *the right ventricle or right auricle* produce grave symptoms, subjective and objective, none of which are here present.

"*Aneurism of the pulmonary artery* does not, I believe, exist in Gunnee; the auscultation-signs, would lie to the left, not to the right of the sternum; there is not a surface-impulse enough for that affection, as commonly observed; the percussion-dulness would lie lower, and to the left; and there would be cyanotic symptoms more or less marked."

"We may, then, I think, venture without misgiving, to assume, as infinitely probable, that Gunnee's aorta has undergone a paulo-post congenital constriction just below the left subclavian. In the first place, we seem almost forced, *per viam exclusionis*, to accept this explanation of his symptoms. Employing little else than the peculiar murmur as an element of inquiry, we find, in truth, that no single one of the ordinary, or the other rarer, forms of cardiac and arterial disease, can fairly be supposed to exist in Gunnee. In the second place, the diagnosis seems plainly warranted on direct grounds. The rhythm, intensity, quality, pitch, site, mode of transmission, and constant identity of characters of the murmur, are all accordant with this idea of its generation; accordant, whether we *à priori* consider, what are likely to be the attributes of a murmur produced by the passage of the blood through a very much constricted spot below the arch; or whether we take the experience derived from cases actually observed. Thus, in two instances which fell under the notice of Blackiston, and which after death were found to be of this kind, a murmur, essentially the same as that heard in Gunnee, had been audible. Next, look to the enlargement and great pulsation of the vessels in the neck ; is not this exactly what we should anticipate to arise as a physiological result of an enduring interference with the aortic current just below the left subclavian ? And is it not, further (in regard of the enlargement, at least), precisely what experience, as illustrated by the diagram, gives us the right to expect ? Again, the slight amount of percussion-dulness, and the sense of impulse across and in the neighbourhood of the upper sternum, may be assigned, with perfect plausibility, to the influence of moderate dilatation of the transverse portion of the arch and of the innominata. Further, consider the singular pulsations and enlarged vessels in the back. The pulsation in two spots near the inferior angles of the scapulæ has all the qualities of aneurismal impulse,—heaving, expansile, forcible. So strong, indeed, is that impulse, that the idea of aneurism of the arch itself, extending backwards, suggests itself for a moment to the mind, and is rejected much rather, because there is no percussion-dulness at the spots, and because, had an aneurism of the arch attained such magnitude, the subjective symptoms must have been of very different gravity and type from those observable in Gunnee, than because the impulse in itself wants the necessary vigour. But this pulsation is evidently caused by dilated and knotty intercostal arteries, in part of their posterior perforating branches ; the vessels can be traced in various other parts of the back of different (but almost everywhere excessive) calibre and impulse. One may look on the knotty tortuous dilatations as so many pseudo-aneurismal sacs. Yet more ; the anomalous distribution of the right ulnar artery tells in favour of the diagnosis ; for an inclination to various arterial anomalies has been noticed in the subjects of this coarctation. Note, too, the rhythm of the murmur—slightly *post*-systolic—just as we should expect of a murmur localized at a certain distance down the aorta, and corresponding to the systole of the left ventricle.

"On the other hand, do no objections to this view of Gunnee's case present themselves ? I do not see any which may not be easily set aside. Thus, on first thought, it may appear, grave functional disturbance must of necessity attend an almost obliterated condition of the thoracic aorta. But so far from experience demonstrating the reality of any such necessity, it does the precise reverse : in some cases of this kind there have actually been no tangible symptoms at all ; in others some such very bearable annoyances as those endured by Gunnee. Again, I confess, one would expect a murmur of the supposed localization to exhibit its maximum force to the left, rather than the right of the sternum. But we cannot feel any surety, in a case of such rarity as this, concerning the modifying conditions of conduction that may exist within the thorax ; and, as actual matter of experience, the murmur has been found loudest to the right of the bone.* Nor can we attach importance to the deficiency of well-marked tactile thrill ; for, though perhaps rather to be looked for, its absence may be accounted for by the smoothness (which experience justifies us in assuming to exist) of the inner surface of the narrowed spot. Nor does the visible and locomotive pulsation of some of Gunnee's arteries shake our faith—in truth, it rather strengthens our conviction—in the justness of the diagnosis. No doubt we are accustomed to refer that physical sign to aortic regurgitation, certain forms of cardiac hypertrophy, and local calcification of the vessels ; but in plain truth, the possible influence of constricted arch has scarcely been thought of by observers,—to so few men does it happen to meet with a case of the kind. The probabilities are, that this affection does produce the sign in question ; for it exists in Gunnee in those vessels originating above the presumed coarctation—is wanting in those springing from any lower point of the aorta. And, lastly, we need not be dismayed by the absence in our patient of certain arterial enlargements (for instance of the epigastric) that have been detected in other instances ; for it would appear there is no absolutely fixed rule, as to which vessels shall undergo dilatation."

838. *Treatment.*—Curative treatment of a case of this stamp is of course out of the question. Nevertheless the part of the

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* In one of Blackiston's cases, at the second right rib ; there was probably an influence produced here by diseased aortic valves,—but the maximum murmur was supra-basic, not basic.

physician is by no means unimportant: symptoms, congestive and other, must be promptly met, as they arise; and the occurrence of such symptoms warded off as far as possible by insisting on a careful system of hygienics,—no matter how true it be, that the subjects of the affection have occasionally lived on in spite of their total disregard. Now of all hygienic rules the most essential is that the patient avoid physical, emotional, and even intellectual excitement,—that his life be a tranquil one. It is of extreme consequence, too, that he escape exposure to cold and damp, and abstain from excesses of all kinds, such as must end in perturbed circulation and local congestions.

II.—ACQUIRED VARIETY.

839. The calibre of the arch of the aorta may undergo diminution both as a result of disease of its own, as also of certain affections of the heart, of the lungs, and of others of the system at large.

(*a*) Under the first head appear contractions depending on exudation-matter connected with the coats of the vessel.

(*b*) Under the second, range themselves reductions of size depending on the closely constrictive action of induration-matter in the pericardium, and on obstruction of the mitral orifice, which entails a deficient supply of blood to the aorta.

(*c*) Next, prolonged obstruction of the pulmonary circulation acts in a somewhat similar way: the calibre of the aorta is below the average in persons cut off with vesicular emphysema of long duration, unless the right ventricle have become the seat of dilated hypertrophy.

(*d*) Lastly, in cancer, phthisis, and probably in tertiary syphilis, the vessel suffers in the same manner, ostensibly from the gradual reduction in the amount of circulating fluid in those diseases.

B.—THORACIC AND ABDOMINAL AORTA.

840. A considerable number of cases are on record in which imperviousness, more or less absolute, of the aorta has been produced in the adult by thrombi forming around prominent spiculæ of calcification, or by the products of inflammation, aided by contraction of the walls of the vessel itself. The lower part of the thoracic and the abdominal aorta are the most common seats of the disease.

841. *Clinical course.*—The course of this affection, symptomatically, may be (*a*) acute or (*b*) chronic.

(*a*) *Acute course.*—In some of the cases belonging to this class, the current of blood, though it may long have been somewhat obstructed, has not become seriously interfered with, until the appearance of acute symptoms,—these symptoms being dyspnœa, anasarca, tendency to gangrene of the lower extremities (evidently obstructive), and hæmoptysis. Still, as the state of the lungs and heart is imperfectly known in the very few recorded instances of the kind, the direct dependence of hæmoptysis on the obstruction must not be too confidently assumed.

But occasionally the attack is to the last degree sudden in its graver manifestations. A merchant, aged twenty-nine, generally enjoying good health, is abruptly, after a few days' indisposition, seized with a burning sensation in the lower extremities, which rises towards the head; he lies almost deprived of consciousness. Shortly after, he is found with a livid bloated countenance, and speechless; the left side almost deprived of motion, the head confused, and the pulse infrequent; when raised, vomiting at once supervenes. After the occurrence of a variety of well-defined local symptoms (stony coldness of surface, lividity, pulselessness of arterial trunks, tenderness of these on pressure, anæsthesia, motor paralysis, mummification of one lower extremity and moist gangrene of the other, the saphæna vein being here also blocked up), all evidently dependent on thrombal and embolic obstruction of the vascular circulation, the patient perished on the 6th of December,—the forty-sixth day from the acute seizure. The abdominal aorta, close to its bifurcation, was completely closed up by a firm, pale red coagulum, tightly adherent to the inner coat, which was smooth and pale. Plastic exudation existed between the membranes; acute inflammation of the arterial texture, as diagnosed during life by Romberg, was the evident cause of the plug-formation. Several other arteries were similarly affected. The difficulty of finding any even remotely probable cause is not the least curious part of this case.*

(*b*) *Chronic course.*—The chronic mode of progress is well illustrated by the following brief particulars of a case which I had the opportunity, during my student-days, of observing in the wards of Louis. In this patient, a female, aged fifty-one, four years previous to death, numbness, first of the right, and, some months later, of the left, lower extremity, were clearly the earliest positive effects of the growing obstruction. These were followed by inability to walk, not from fatigue or cardiac suffering, but from pain, coldness, and increasing numbness in the legs. Subsequently, the effects of organic disease of the heart (she had mitral constriction, dilated hypertrophy of the right ventricle, palpitation, pulmonary apoplexy, and hæmoptysis) threw those of the aortic obstruction into the shade, and eventually cut her off in a few days after admission to the hospital. Twice in the course of her illness she had slight and passing œdema of the lower extremities, but there was none on her admission, and at death scarcely any. Besides a complete local obliteration above the iliacs, the calibre of the aorta generally, and of its branches, was below the average in this woman. No attempt appeared to have been made at anastomotic enlargements; the disease had, to all seeming, originated in inflammation of the vessel.

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* Romberg, Dis. of Nervous System, vol. ii., p. 238.

Gull records a very remarkable case, in which, obstruction of the abdominal portion of the vessel having produced serious paraplegic symptoms; eventually, in proportion as anastomotic enlargements ensued, the motor paralysis materially improved.*

§ VII.—RUPTURES, PERFORATIONS, AND INJURIES OF THE AORTA.

842. Complete rupture of the aorta has sometimes occurred, where, if observers may be implicitly trusted, the vessel was free from any form of pre-existing disease; more commonly more or less extensive calcification has been found; in rarer instances a softened state of the coats is referred to in descriptions.

843. (*a*) *Spontaneous.*—Rupture appears particularly prone to occur in the ascending portion of the thoracic aorta. More frequently the fissure is found at right angles with the long axis of the vessel than in any other direction.

844. Of thirteen cases belonging to this category, eight were furnished by males, five by females.† Spontaneous rupture seems to be most frequent between the ages of thirty-five and fifty.

845. Death is not necessarily instantaneous; the external coat may not give way at first, and some hours, or even days may elapse, before a second rupture, breaking through this also, destroys life instanter.

846. (*b*) *Perforative.*—Ulcerative perforation of the vessel has in rare instances been observed,—commonly as the result of a foreign body making its way from the œsophagus.‡

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* Guy's Hosp. Reports, 1857.

† Crisp, Dis. of Arteries, p. 289.

‡ Arch. Gén. Méd. 1825; Flower, Lancet, June, 1853.

847. Ruptures from external violence of various kinds are of rare occurrence, and almost limited to the male sex,—probably because more exposed to their direct causes. More or less serious evidences of injury are, as a rule, to be found, simultaneously, on the thoracic surface.

But a question of great interest is, to what extent physical shocks to the surface (especially about the second right cartilage, where the vessel most closely nears the parietes, and also especially in thin people) may inflict injury on the artery, without leaving any very notable trace on the integuments? I saw a case some years ago raising this question in an urgent form, in connection with a railway concussion;* and the inquiries made on that occasion revealed a total absence of fixed professional opinion, as well as extreme poverty of printed information, bearing on the matter. Nor can I, to my regret, help in supplying the deficiency, except by venturing to direct attention to the subject. Here are the facts. A man, aged under forty (younger, consequently, than the average of sufferers from aneurism of the aorta), was to all seeming in perfect health, when he was thrown violently forward by a sudden jar of the train in which he was seated,—the upper part of the right side of the chest pitching against an arm-rest between two seats opposite. It was deposed in evidence that this person had never complained of chest-symptoms, and that he had not consulted any medical man concerning the state of his heart or lungs. Now, within two hours after the accident the medical officer of the railway company concerned found a pulsating prominence in the neighbourhood of the second right cartilage. This aneurism, for aneurism it was, eventually destroyed the sufferer in about eleven months after its discovery. The man pleaded that the aneurism was caused by the injury. The following arguments might be used in favour of his plea. 1. The chest-wall, where flexible, and in thin persons (among whom he ranked), is so easily depressed, that, as is well known, murmur may be produced by a very moderate amount of stethoscopic pressure. 2. The injuries to the thorax, which have produced actual rupture of the aorta, have ranged between violences the most terrible and others so trivial, as to appear incapable of producing the effect. 3. Injuries to the chest-wall have occasionally ruptured the lung, without either fracture of a rib, or serious external marks of violence.† 4. Morgagni records several cases in which abdominal organs were torn by blows from sticks, though there was no injury to the skin, not even an ecchymosed stain.* 5. Numerous instances have been published in which a direct connection between external injury and aneurism of the arch has been traced and admitted.† 6. A case is recorded in which an aneurism was first perceived within twenty-four hours after the receipt of a blow with the fist on the right side of the chest.‡

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* Case seen in consultation with Skey and Erichsen.

† Art. Emphysema, Cyc. of Surgery, p. 82.

Now conceding that all these arguments put together do not constitute proof in the affirmative, they show that it would be difficult enough to maintain a negative. It seems to me well conceivable, that when calcification has pre-existed to any extent in the vessel, an injury of the kind described might lead to sudden breakage of the lining and middle, and blood-distension of the external, tunics.

SECTION II.—CORONARY VESSELS.

§ I.—RUPTURE OF A CORONARY ARTERY.

848. An individual, subject to palpitation, had a sudden attack of retching, with cardiac anguish, and died in an hour. The left coronary artery, widened, hardened, and fragile, was broken across from the aorta; effusion of blood had taken place between the pericardium and great vessels.§

§ II.—ANEURISM OF A CORONARY ARTERY.

849. This is a very rare affection in the sacculated form; while fusiform dilatations of the origin of the vessel, and widening of its general tract, are far from rare.

850. The sac in the few instances on record has been included in the substance of the ventricle or of the auricle (almost always on the left side). It has contained thrombi, sometimes distinctly laminated and partially decolourised.‖

851. In some of these few cases death has actually resulted from, or been hastened by, rupture of the sac: where no rupture has occurred, there is a want of evidence that the affection gave rise

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* De Sed. et Caus. Morb. Ep. liv. § 15.

† *E. g.*, Cases vii. x. xi. xii., in Crisp's collection, p. 150, *et seq.*

‡ Case vii. Crisp, *loc. cit.*

§ Lombard. Gaz. Méd. de Paris, iii., 644.

‖ Peacock, On Aneurism of the Coronary Artery, p. 5.

to any symptom. It is noteworthy that in no single instance, though in some cases associated with calcification and general dilatation of the vessel, has aneurism of the coronary artery been said to have caused symptoms of angina pectoris,—a fact which would almost require me to modify somewhat a statement, hazarded concerning the influence of the coronary arteries, in the history of the latter disease [341].

§ III.—VARIX OF THE CORONARY VEINS.

852. Sudden death has been occasionally caused by rupture of a varicose coronary vein. In two cases of the kind, observed by Albers, there had been symptoms of "cardiac asthma," but evidently traceable rather to the state of the heart itself, than of the dilated vein.*

SECTION III.—PULMONARY ARTERY.

§ I.—THROMBOSIS OF THE PULMONARY ARTERY.

853. The pulmonary artery, like all the channels in the body for venous blood, is prone to suffer obstruction on a small or large scale from the coagulation of its contained fluid.

854. *Causes.*—The conditions leading to this coagulation are very similar to those traceable in the analogous case of thrombosis within the heart's cavities [735]. They may be arranged as follows: (*a*) blood-alteration, of which the essential tendency is to increase the coagulating property of the fluid; (*b*) some form of constitutional state rendering the circulation more sluggish than natural; (*c*) local causes of obstruction within the vessel concerned.

(*a*) The blood-conditions are, especially, hyperinosis, the peculiar crasis in parturient women, and spanæmia.

(*b*) All forms of debility, whether traceable directly or not to the influence of disease, past or present, fall under this head. In the convalescence of acute diseases, particularly those which present other favouring conditions, it is probable slight coagulation may happen pretty often, while it is certain that the process occurs on a serious scale in a small proportion of instances. Pneumonia supplies an illustration.† There is, I think, motive for believing that, "relapse" is occasionally constituted, not by real return of the disease, but by spontaneous thrombosis within certain branches of the pulmonary artery; while, it seems to me, there can be no reasonable doubt, protracted convalescence, and long continuance of dulness under percussion, with some form of bronchial breathing, are sometimes due to such coagulation. Syncopal tendency, as is familiarly known, is another condition which dynamically promotes the clotting-process.

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* Brit. and For. Med. Rev., July, 1845.

† Louis, in Baron, *loc. cit.*, p. 17.

(*c*) The local aids vary with the position in the vessel, where the deposition of the fibrine takes place. In one class of cases the minute branches are its primary seat, whence it gradually extends to the larger trunks. Here, either pressure-obstruction, on the outside, by nodulous blood-infiltration, by exudation-matter, by adventitious deposits, and by infiltrating growths,—or obstruction by an embolic process within the minute ramusculi, lays the groundwork. The embolic or plugging agent may, in the latter case, be derived from materials, fibrinous or exudative, borne along from the right side of the heart directly, or be furnished by minute fragments, separated from thrombi formed in some distant part of the venous system, and carried along until they reach portions of the vessel too narrow to allow of further onward movement. This is a mechanism rendered, *à priori*, probable by the special minuteness of the pulmonary capillaries, as well as by the experiments of Cruveilhier and others, which long since showed that various foreign bodies introduced into the venous current stagnate in the minutest vessels of the lung. This mode of production, however, can obviously play no part in cases where, on the one hand, there is no evidence of coagulation in any other, near or distant, part of the venous system; and where, on the other, the clotting process is limited to the larger branches of the pulmonary artery. In this form of case, where the larger branches are alone implicated, local aid to stagnation is found in the abruptness with which the artery divides into a number of branches, and in the numerous angular projections into the current at their points of bifurcation.*

Although disease of the inner coat of the pulmonary artery, inflammatory or other, may act, and has occasionally acted, as the local cause of obstructed current, yet instances of the kind are singularly rare; nothing can be more clearly demonstrated in the entire group of cases of venous stagnation and thrombosis than the habitual absence and consequent non-necessity of primary vascular inflammation.

855. *Anatomical appearances.*—(*a*) Pulmonary thrombi are

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* Humphry on Venous Coagulation, p. 29.

either uniformly fibrinous looking on the surface, or of variously mottled colours and appearances,—arising out of mixture of hæmatine and of changes of tint which this has undergone. They may be almost black on the surface; generally speaking firm, sometimes remarkably so, and smooth on the outside, they are usually short,—not reaching in the large branches beyond the ramification immediately next to that in which they mainly formed. Clots may coexist in various parts of the vessel,—several independent sources of coagulation being thus simultaneously, or nearly so, at work. The distal end of the thrombus is generally more or less fined off; but this appearance may be, and often is, wanting in small sized coagula,—which terminate abruptly and, as it were, shapelessly. The clot is generally adherent; sometimes throughout its whole length and periphery, more commonly by a limited spot only. The adhesion may be sufficiently firm to cause distinct difficulty in removal; so that the lining membrane undergoes some very slight superficial injury in the operation.

(*b*) Provided the patient be not at once destroyed either by the clotting process mainly, or by this added to pre-existing disease, various changes may occur in the thrombi, more or less identical with those observed in other parts of the venous system. Central softening may take place; and probably prelude disintegration and more or less effective solution and dispersion. Or the coagula, growing firmly to the wall of the vessel, may harden, and in this condition undergo more or less complete absorption, leaving behind them various residues, filamentous, lumpy, or stratiform. There seems reason to believe, that some at least of so-called stony concretions in the lungs have been nothing more than phlebolithic transformations of thrombi within the pulmonary artery.

(*c*) In the small branches complete and permanent obliteration may occur.

(*d*) Sequential mischief to the coats of the vessel is not observed; no vascularity of the lining, nor exudation into the external, membrane. At least this is very certainly the rule; but that in some rare instances of death, not really obstructive and of unintelligible mechanism, the fatal event may have depended on sequential pulmonary arteritis, seems, however unproven, by no means unlikely.

856. *Symptoms.*—The symptoms vary extremely with the site, the size and the rapidity of formation of the coagula. In this

clinical point of view cases may be conveniently thrown into three groups.

857. *First group.*—When the clotting process is on a limited scale and occurs within branches of small calibre, no definite symptoms can, practically speaking, arise. At least the remnants of thrombi, evidently of old formation, have occasionally been found, where the lungs had not been suspected to be the seat of any long-past mischief.

858. I am not aware that the existence of such old-standing obstructions in the branches of the pulmonary artery has ever been diagnosed; but I once saw a case, presenting the following peculiarities, which possibly might be explained on such hypothesis. A middle-aged man, suffering from dyspnœa and chronic cough, free from the ordinary signs and symptoms of chronic consolidation, emphysema, tubercle, bronchitis and the graver class of pulmonary and cardiac diseases, presented a strong blowing murmur, following the direction of the pulmonary artery, on the left of the sternum, inaudible at the apex and also at the base of the heart, extending well across and into the left axilla and to the costal angles, but not to the actual vertebral groove. On three examinations, at intervals of about a fortnight, this murmur presented precisely the same characters. I could not divest myself of the idea that it probably originated about the main branches of the left pulmonary artery,—and if so (as there was no condition present to cause pressure on the vessel from without) partial internal obstruction seemed to afford the most likely explanation.

859. *Second group.*—In a second class of cases the amount of coagulation is considerable; while the process, slow at first, eventually, and with variable abruptness, rapidly advances. As might be expected, this mode of progress is reproduced in the course of the symptoms.

860. *Symptoms.*—The essential effect is dyspnœa, more or less marked, but commonly intense, the patient crying out for air; to this are added prostration, faintness, or actual syncope; tumultuous movement of the heart, or action scarcely differing from the usual type; pulse at the wrist feeble to actual deficiency almost; if venesection be performed, scarcely any blood flows from the vessel; sense of tightness at the præcordia; turgid lividity of the face; œdema of the extremities, with partial violet discoloration of their surface; great general distress, jactitation and dread of dissolution, while the encephalic functions remain unaffected.

861. *Diagnosis.*—Total absence of ordinary acute cardiac and pulmonary affections may be satisfactorily substantiated, provided the patient's condition permits of the necessary examination being made. In some cases (but, as *post-mortem* examinations prove, not in all) the physical signs of accumulation in the right cavities of the heart may be expected to occur [414, 742]. The only physical evidence connected with the vessel itself would probably be systolic basic murmur, following the course of the pulmonary main trunk, and of its immediate divisions to the left and the right of the sternum. This sign I most certainly heard in the case of an aged gentleman, whose life was brought to a sudden close, in the course of an acute affection, by coagulation in the pulmonary artery, and, to a moderate extent, in the right ventricle. The murmur in question can, of course, be only expected to exist where the large trunks are involved; of physical evidence of deep-seated acute coagulation in the small branches I know nothing by experience. Perhaps some such murmur, as that described a moment since from the history of a chronic case, might be detected [858].

862. *Course.*—In almost all carefully recorded cases, belonging to this category, notable relief from the first anguish has been experienced; recurrence, more than once, of paroxysmal suffocation having taken place before the final struggle.

It appears, then, that the influence of coagulation in the pulmonary artery on life varies exceedingly. Not only may the formation of clots, even of good size, in the larger branches not destroy life at the time, but they may not sensibly shorten existence. Or such formation may bring life to a very sudden, though not actually instantaneous, close, out of its unaided influence; or it may, in a variety of morbid states, hasten an issue, which but for it might, perhaps, have been indefinitely averted.

863. *Prognosis.*—Under all circumstances, were the diagnosis established, the prognosis becomes to the last degree grave: we have no right to expect that in any particular instance the course of events shall place the patient in the exceptional category of those, in whom the site and relationships of the coagula save them from fatal results.

864. *Treatment.*—The intense turgidness of the external venous system suggests the advantage of relieving the stasis by blood-letting; but when venesection has been tried, no such relief has been obtained—in truth, the blood has not flowed with freedom. The probabilities are, too, that anything like a copious flow, which

certainly might lessen the evils of venous plethora for the moment, would eventually place the patient in, if possible, a worse position than before. In cases running a lingering course, dry cupping of the chest, the application of Junod's boot to a limb, mustard poultices to various parts of the surface, with free use of diffusible and alcoholic stimuli, afford the best chance of maintaining the circulation. If rally occur from the first urgent effects, the management of the case should be conducted in the manner recommended for coagulation within the heart [747].

865. *Third group.*—A third class of cases is typically represented by the following brief narrative :—

"A healthy female, aged 38, naturally confined of a fourth healthy child (lochia moderate, milk-secretion artificially stopped, without inconvenience of any kind), appears in the most satisfactory condition possible on the thirteenth day after delivery. After a good night's rest, she makes a light breakfast in bed on the fourteenth day, spontaneously remarking, how thoroughly well she feels. She then rises (for the first time), chats with people around her, while her hair is being dressed ; when suddenly, without warning of any kind, she cries out 'Oh, my God ;' her head falls on one side, and she instantly expires.

"Sectio cadaveris the following day ; weather cold. No appearance of putrefaction ; cadaveric staining posteriorly. Encephalon strikingly pale, its arteries empty. Lungs remarkably healthy-looking. Heart small ; no notable sub-pericardial fat ; from right cavities a large quantity of blood escapes, coagulated to various amounts, without homogeneous or laminated clot,—that in the auricle being of the consistence of partially melted jelly, that in the ventricle (reaching somewhat into the pulmonary artery) being somewhat firmer. Aorta empty, calibre below usual par, healthy. Heart's tissue somewhat flaccid right side, contracted firmly left ; carefully examined, microscopically, is found perfectly free from fatty metamorphosis.

"The contents of the thorax being next removed, very fluid blood pours in abundance from the venæ cavæ,—a state of fluidity which, with one exception, was strikingly evident throughout the entire venous system. *Right pulmonary artery empty ;* left contains coagula of gradually increasing consistence as the branches become smaller : thus, in the fourth or fifth divisions, the clots are thin, resisting, elastic, but coloured shreds. The periphery of the clots, which are non-laminated in the larger branches, is firmer than their centre ; nowhere can a central nucleus be found. Uterus firm, size of a large fist ; the veins in its substance contain but little blood ; those passing out of it, on the contrary, are distended by very slightly clotted blood, like that in the right auricle, as are also the inferior cava, the iliacs, and the femorals, as far as the external saphena ; but the most careful search fails to detect any particle of fibrinous coagulum."

These particulars, very kindly furnished me by Dr. Guéneau de Mussy, typify a class of cases, of which a considerable number have been observed among females of late years. The essential features of the group are these : recent parturition (generally not a fortnight past) ; natural and healthy progress after delivery ; slight effort ; posture sitting or standing ; sudden cry, unpreceded by the slightest uneasiness of any kind, followed by instantaneous death,—so instantaneous in some cases as to be assimilable to that produced by section of the medulla oblongata. The body is opened ; and nothing found to attract attention except coagula in the pulmonary artery, associated or not with coagula in other portions of the venous system.

866. The interpretation of the facts, commonly adopted, ascribes the fatal event to abrupt stoppage of circulation in the lungs, by the clotting-process in their arteries. That this process actually commences in the large branches or trunk is held by some observers; while others maintain the plugging theory,—the necessary fibrinous plugs for the minute vessels being furnished by coagula in some distant vein.

The question of precĕdence in the sites of coagulation is of altogether subsidiary importance; yet it may be as well to observe, that careful examination has failed in several instances to detect any formed fibrinous coagulum within the veins of sufficient tenacity to justify the belief, that fragments would have borne the journey through an extensive venous tract, without reduction to a state of deliquescence and fluidity. Independent clotting within the pulmonary artery is in such cases the only legitimate hypothesis.

But, to come to the essential matter, Wherein lies the proof that the clotting-process is really the *cause*, instead of being merely a *concomitant*, of death in these terrible cases? In several instances of the kind the coagula have been found in medium-sized and small branches only,—they have not filled the bore of the vessel,—they have even (as in the instance just narrated) been limited to the artery of one lung. It can readily be conceived, as already shown, that death may be effected, and rapidly effected, by the formation of clots in the pulmonary main trunks. But even then instantaneous death is not habitually to be looked for,—the formation of such clots cannot be itself the work of an instant of time. Does it not seem most rational to look upon these deaths as primarily and essentially syncopal,—the clotting-process being in some cases a possible coincidence, in the majority a sequence, and in all promoted by the plastic crasis of the blood in ex-parturient women? The immediate manner of death points emphatically to syncope; and further the brain is pale, its arteries empty: Is this the state to be expected, had death really arisen from sudden arrest in the supply of blood to the pulmonary arterial rete?[*] There is no reason to assume, still less any evidence to prove, that stoppage of circulation through the main pulmonary trunk would *instantaneously* exercise such reflex paralytic influence on the left ventricle, as to prevent it from emptying itself of any blood, that might be within its cavity at the instant of that stoppage.

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[*] Just as in ordinary syncope from heart-failure; or as from *sudden* crushing of the encephalon in experiments on animals.

The causes of the syncope, in turn, lie beyond the pale of positive demonstration. Awaiting this, speculation may be indulged in. Now in the greater number of cases the fatal event has occurred in the erect or sitting posture, oftentimes immediately upon some emotional excitement; while further, in more than one instance, the victim has suffered for a greater or less length of time from mental disquietude and anxiety. In some cases of the class thrombal obstruction of the coronary arteries may possibly play its part in bringing life to an abrupt close; but, either for or against this suggestion, evidence is wanting.

867. There is commonly no time either for *diagnosis* or for *treatment*. In point of fact, the patient has usually been either actually, or well-nigh, dead, when medical aid has been obtained. Should life not be extinct, when the patient is seen, the treatment for syncope is clearly indicated. Ammonia, administered by the mouth (if this be impossible, *per rectum*), is the drug most likely to prove beneficial, if time be given for the use of remedies.

§ II. INFLAMMATION OF THE PULMONARY ARTERY.

868. We have seen that in cases of coagulation within the pulmonary artery, as a class, inflammation of the coats of the vessel is very rare. It is rare not only as a primary condition, but even in that sequential form, which pretty frequently follows on the clotting-process, when occurring in the veins of the extremities. And further it appears very distinctly, that authors, who have insisted much on the comparative frequency of inflammation of the pulmonary artery, have assumed its existence wherever firm fibrinous coagula were found, especially if associated with sanguineous discoloration of the vessel.*

But such coagula and such discoloration by no means prove that inflammation has occurred;—*post-mortem* staining has evidently been confounded with true hyperæmic injection. In a word, examples of genuine inflammation, demonstrated by lymph-exudation within the tunics of the vessel, or on its inner surface, with fibrinous dropsy about the external coat, are very difficult to discover. These conditions, combined with capillary sub-epithelial injection, roughness and loss of polish of the lining membrane, constitute the anatomical characters of the disease.

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* The cases of Cruveilhier (Anat. Path. livr. xi.), often quoted as examples of inflammation of the vessel, sequential to uterine phlebitis after delivery, possess no proven claims to rank in the category of inflammations. The central "pus" in the clots was obviously softened fibrine.

These appearances may be found in certain divisions of the main trunk;[*] or they may be limited to the valves, as in an instance recorded by Graves.[†]

869. Running an acute or sub-acute course, this inflammation is alleged to have been observed especially in pyæmia and certain other diseased conditions of the blood, in pneumonia, as a sequence of phlebitis, uterine or other, in "Bright's disease," and as an idiopathic (?) state. But the actual evidence, tendered of its existence, is often anything but convincing,—as may be easily ascertained by perusing the essay of Chevers.[‡]

870. *Physical signs.*—No attempt has been made, so far as I know, to establish a definite physical history of the disease,—and never having seen a satisfactory case of the kind, I am unable to supply the deficiency. The probabilities obviously are, that physical signs would arise rather out of the clotting-process attending the inflammation, than out of the changes in the vessel itself.

871. *Symptoms.*—So, too, the symptoms in alleged inflammation have been identically the same as those arising as a consequence of rapid, but not instantaneous, coagulation, without the intervention of an inflammatory process. To the outline of these symptoms, already given, the reader may consequently be referred [860].

872. *Treatment.*—Treatment of inflammation of the pulmonary artery cannot be said to have ever been even attempted,—seeing that the affection has never been diagnosed. And in as much as it forms an addition only to some profoundly serious pre-existent complaint, active measures can hardly be conceived warrantable. Were the diagnosis accomplished, counter-irritation and dry cupping of the chest, in association with the internal remedies advised in cases of coagulation, ought to be tried. The tendency to sinking calls for free exhibition of stimuli.

§ III.—ULCERATION OF THE PULMONARY ARTERY.

873. The rarity of ulcerative perforation of the pulmonary artery, on anything like a large scale, in the course of phthisis, is familiarly known. The branches of the vessel amid tuberculous deposit become clogged with coagula, so that even if a branch of

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[*] L'Expérience, Mars, 1842.
[†] Dublin Journal, vol. xxii.
[‡] Morbid Conditions of the Pulmonary Artery, p. 79 *et seq.*

some size undergo perforation, no hæmorrhage ensues. The demonstration of the complete obliteration of the finer divisions of the vessel, amid and around tuberculous deposit, given by S. van der Kolk and Guillot, shows in a very satisfactory manner how it happens, that destruction of wall may occur even extensively without giving rise to clinical disturbance.[*]

Still, both in the course of ordinary phthisis and of tuberculization of the bronchial glands, fatal hæmorrhage may, and does every now and then, arise from perforation of a large branch, or, in the latter case, of the trunk, of the pulmonary artery.

874. In rare instances of abscess, and in more frequent ones of gangrene of the lung, a branch of some size, unprotected by coagula, is partially destroyed. The coagulating process seems to occur more sluggishly in the child than in the adult; and hence it is that hæmoptysis is relatively more common in childhood (a period at which its occurrence from all causes combined is, absolutely speaking, so rare), than after puberty, as an effect of sphacelated lung.

§ IV.—RUPTURE OF THE PULMONARY ARTERY AND ITS MAIN BRANCHES.

875. (*a*) Rupture of the *main trunk* has been observed, as in the aorta, both from emotional excitement, and under the influence of violent effort, whether the vessel were actually affected by, or free from, well-defined previous disease of its coats.[†]

876. Although death has been described as sudden in these cases, it does not appear to have been actually instantaneous in any of them. Prescott Hewett relates a case in which, though the vessel was extensively ruptured, and the left auricle lacerated, the individual survived nearly four hours.[‡]

877. (*b*) And, where a *main branch* only is concerned, life may be much longer in ebbing away. A man, aged sixty-three, long the subject of bronchitis, the signs of which were alone discovered by auscultation, had a sudden attack of violent hæmoptysis. The bleeding, at first controlled, recurred, and death followed from exhaustion on the fourth day; the right pulmonary artery had undergone rupture just at its entrance into the lung; there

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[*] Louis on Phthisis, Syd. Soc. Trans., p. 29.

[†] Chevers' essay contains examples of all these modes of occurrence.

[‡] Lond. Med. Gaz., 1847.

were no tubercles or other new product in the tissue of the organ.*

On the other hand, death may be instantaneous, as in the case of a soldier, aged thirty-four, recorded by A. Mackay. Here the rent, about a quarter of an inch long, was surrounded by a "chronic ulcer" about the size of a sixpence.†

§ V.—GENERAL DILATATION.

878. General moderate dilatation of the pulmonary artery, trunk, and branches, is occasionally to be seen in connection with dilated hypertrophy of the right ventricle, sequential or not,‡ to long-standing vesicular emphysema. Widening of the vessel sometimes coexists with thickening of its walls, as an effect of the labour of the pulmonary circulation caused by mitral disease. The strong accentuation of the second sound at the left second cartilage, in comparison with the corresponding right cartilage, as originally shown by Skoda, will, in some, but by no means in all, instances, guide to the detection of this hypertrophous widening (p. 93).

879. The normal tendency of the mass of the lungs to decrease with advancing age, insomuch that to undergo a genuine senile atrophy seems part of their physiological destiny, would lead us to anticipate, as a probability, that, unlike the aorta, the pulmonary artery may rather diminish than increase in calibre as years roll on. But this anticipation is not borne out by the fact. Very certainly the width of the vessel seems to incrêase in advanced life. Possibly this may depend on the great frequency of bronchitic disease.

880. *Diagnosis.*—I do not know of any signs or symptoms whereby this condition might be diagnosticated. Skoda, speaking of uniform widening of the vessel as a frequent state, observes he has not known it carried sufficiently far to deaden the percussion-sound. Stokes gives the prominent features of a case where the dilatation was so great as to render the valves incompetent,—but there were, unluckily, so many other associated morbid states in the heart and aorta, that the interesting narrative practically contributes nothing to future diagnosis. A curious effect of local dilatation, coexisting with tricuspid reflux, has already been referred to [617].

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* Fearn, Provincial Journal, 1845.

† Med. Times, Oct. 18, 1862.

‡ Kernis, U. C. H., Females, vol. ii. p. 240.

§ VI.—ANEURISM.

I.—SACCULATED VARIETY.

881. Sacculated aneurism of the pulmonary artery, a very rare disease, may dilate the vessel to the size of a goose-egg and upwards. The sac may be simple or compound (Diag. p. 476, figs. 3 and 4); and, as in a case observed by Skoda, accompanied with extreme narrowing of both the main branches. Again, the valves may be natural or contracted. The aorta has been either normal or slightly dilated, or dilated below, and then contracted near the ductus arteriosus. The left ventricle may be natural; the right has been dilated and hypertrophous. In one case the ventricles communicated by a permanent aperture.* Bronchitis, pneumonia, and hydrothorax, occasionally prove sequential states.

882. *Physical signs.*—The physical signs in indubitable cases have somewhat varied. The most satisfactory, actually observed, have been pulsating prominence, centralising in the second left interspace, close to the sternum, and the seat of strong systolic thrill; and systolic, superficial, loud, harsh, rasping or grating murmur, of maximum force at the prominence, and followed, in one instance, where a pulmonary valve was contracted, by a very short diastolic murmur. But in Skoda's case there is not a single sign mentioned fairly referrible to the aneurism; he himself supposes the absence of murmur explicable by the narrowness of the two main divisions of the vessel.

883. *Symptoms.*—The prominent symptomatic states have been lividity of face, described as of cyanotic depth of tint in some cases, anasarca, ascites, great dyspnœa, cough and frequently recurring bronchitis, scanty urine, suppression of the catamenia, cephalalgia, and pain in the chest or at the epigastrium. The pulse, of variable frequency, is without special character.

But the most apparently significant symptom of the series may be wanting: Fletcher's patient was pale instead of being livid, though the two ventricles communicated besides.

884. Pulmonary aneurism has been observed in both sexes; in persons in the prime of life, and in girls under twenty.

885. Information is yet wanting as to the duration of life. In Skoda's case death followed the appearance of dropsy in three months; Fletcher's patient lived certainly twenty months—probably three years—with the disease.

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* Fletcher, Med. Chir. Trans., vol. xxv.

886. *Diagnosis*—The diagnosis of aneurism of this vessel may be considered tolerably certain, if pulsating prominence, limited to the left of the sternum and the second and third cartilages, furnishing strong, superficial, systolic thrill, and harsh murmur of the same rhythm—neither of them discoverable at the top of the sternum or above the clavicles—coexist with deep lividity of the face and anasarca, for which symptoms no ordinary explanation can be found. But aneurism of the ascending and transverse part of the arch of the aorta may protrude mainly to the left of the sternum [780 (*a*)]; and tumour in the mediastinum, pressing on, and receiving impulse from, the pulmonary artery, as also great hypertrophy of the left auricle, may simulate more or less closely these states; lastly, pulsating empyema must not be altogether forgotten.

II.—DISSECTING VARIETY.

887. A soldier, in the pontoon service, aged twenty-one years, well able to discharge his laborious duties during the three months he had been in the army, in spite of slight dyspnœa, went to bed in good health, December 5th, having been employed in carrying wood during the day. In the night he was roused by violent pain to the right of the sternum, with great dyspnœa; all this subsided in a few minutes, leaving him pale, cold, anxious, and perspiring. In a short time nothing remained but præcordial uneasiness, slight palpitation, and feebleness of voice. Next day he seemed well, but at night complained of exhaustion; the face became pale, the surface cold, the pulse imperceptible, and in a very short time he expired. The pericardium contained a large quantity of coagulated blood; there were extensive calcareous concretions in the pulmonary artery, the inner coat of the vessel being separated from the elastic for the space of three inches from its union with the right ventricle, and torn into shreds, projecting into the cavity of the vessel, while further on the lining membrane was coated with fibrine. Just at the origin of the vessel appeared a hole the size of a fourpenny piece, through which the escape into the pericardium had occurred.*

III.—VARIETY AFFECTING MINUTE BRANCHES.

888. Rasmussen has shown that hæmoptysis in the advanced condition of tuberculous disintegration of the lungs is, in a certain

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* Helmbrecht, Caspar's Wochenscrift, March, 1842.

proportion of cases at least, due to rupture of aneurismal dilatations of the small branches of the pulmonary artery coasting the walls of cavities.*

The earliest recorded example of this mode of production of tuberculous pulmonary hæmorrhage is, as far as I know, that by S. W. Fearn. Death took place by "venous-looking" hæmorrhages, frequently recurring, and at length suffocating the patient. A burst aneurismal sac, as large as a nutmeg, formed on a branch of the pulmonary artery the size of a crow-quill, jutted into an empty cavity.†

§ VII.—ATHEROMA AND CALCIFICATION.

889. That atheroma of the pulmonary artery, more especially of its medium-sized divisions, is not infinitely rare, has been fully ascertained, since the habit of slitting up the vessel in ordinary *post-mortem* examinations has grown common. But, except as affording a nidus for calcareous precipitation, this fatty deposit seldom does any demonstrable mischief. I cannot help suspecting strongly, however, that in some instances it aids in the production of pulmonary apoplexy; and to one indubitable case, where it was connected with profuse and repeated hæmoptysis, otherwise inexplicable, I have referred in another place.‡ Calcification, certainly very rare, is generally associated (probably, in some measure, at least, as an effect) with hypertrophy of the right ventricle. Of its perilous character, when locally carried to an extreme amount, the case just related affords sufficient proof [887].

§ VIII.—CANCER.

890. In cases of cancer of the lung-substance, especially of the encephaloid species, it is not uncommon to find more or less extensive plugging of even large-sized branches of the pulmonary artery with coagulated fibrine and cancerous substance. The mechanism of the occurrence seems to be this: obstruction to the blood-current is first effected in minute branches by pressure from without of the cancerous substance infiltrating the adjoining parenchyma; next, the stagnating blood coagulates; thirdly, the development of cancerous blastema into cell-structure takes place

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* On Hæmoptysis, Transl. from Danish by Moore, Ed. Med. Journ., 1868.

† Lancet, Feb. 6, 1841.

‡ Diseases of the Lungs, 4th ed. p. 400.

within the meshes of the resulting coagula. As I have searched in vain for cancer-cells, and even for suspicious molecular matter in actual circulation with the general mass of the moving blood in cases of well-developed general cancerous contamination, and as other observers have not proved more successful, I cannot suppose that the appearance of firm encephaloid in the pulmonary artery can be due to the mere evolution of ready-formed solid elements entangled in the stagnating fluid. Nor, on the other hand, do I know of any evidence proving, or even tending to show, that perforation of the vessel by cancerous growth on its exterior, and subsequent vegetation in its interior, ever occurs, after the fashion frequently observed in the case of the bronchia.

891. The narrative, apparently most strongly militating in favour of actual translation of cancer-cells through the general venous circulation, that I know of, has been reported by A. Wernher. A man, aged twenty-two, first seen January 22, with a rapidly-growing encephaloid mass of the knee and tibia, is attacked on the 27th with sudden præcordial pain to the left, then to the right, of the sternum, without cough, palpitation, increased cardiac dulness, or murmur; pulse 140. On the following night several attacks of dyspnœa occurred, with scarcely perceptible pulse; on the third and fourth days, both, some pure coagulated blood was expectorated. Two or three days later the dyspnœa diminished, and the man appeared very much as before his acute seizure. Amputation was performed February 7th. Pyrexia, hæmoptysis, prostration followed; on the 19th rigors, dulness at the posterior bases; and on the 24th death from asphyxia and collapse. The iliac and crural veins, and their branches, as well as the cava, seemed normal, and free from coagula. The right pulmonary artery was almost filled with coherent cancerous masses; the left similarly obstructed, but to a less degree. In some spots the cancerous substance was incorporated with the vascular walls, though generally those walls proved perfectly healthy. The capillaries and pulmonary veins were quite free. The cancerous masses consisted almost throughout of large oval primary cells, with single or double nucleus, like those of the encephaloid in the leg. In the blood of the right heart, and of the ascending cava, similar cells were found; but none of the kind in any other blood. The lung-substance had been destroyed by gangrene in some parts, of which the pulmonary arterial branches had specially undergone obliteration.*

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* Henle's Zeitschrift, Neue Folge, 5r. Bd., p. 109, 1854.

It must be conceded the discovery of formed cancer-cells in the blood of the right heart and cava inferior, and of these parts alone, carries with it serious significance as to the possibility, without, perhaps, positively demonstrating the reality, of actual cell-translation. Let me note, in passing, how distinctly the clinical history of the case strengthens the statement made in the account of pulmonary blood-concretions, as to the improvement which invariably takes place in cases of coagulative obstruction after the first urgent suffering [859]. As far as it goes, the narrative supports the view, that the mode of death from pulmonary obstruction should be asphyxial and not syncopal [866]; but obviously the case is not assimilable to one of abrupt thrombal obstruction of the main pulmonary trunk.

SECTION IV.—PULMONARY VEINS.

§ I.—INFLAMMATION.

892. Information is yet more deficient on the subject of inflammation of the pulmonary veins than of the pulmonary arteries.

893. R. Lee records a case, of which the following are the more essential particulars. A primiparous woman, aged twenty, is seized with rigors on the fifth day after delivery: these are followed by rapid pulse and dyspnœa with left thoracic and abdominal pain. Death occurred on the twenty-fourth day of the seizure. Both pleuræ and both lungs were inflamed. The trunk of the left pulmonary vein, near its entrance into the left auricle, was found plugged with a greenish, soft, firmly adherent clot of lymph, extending into several of the principal branches. The lymph-clot appeared brightly scarlet on its outer surface when separated from the vein. The smaller branches, into which the solid lymph did not reach, were filled with pus [softened fibrine?], and in some parts coated with a delicate layer of yellow lymph.* A very similar case has been described by Adams.† Singularly enough, neither one nor the other of these observers adverts in any way to the condition of the pulmonary artery.

894. These narratives furnish no clue to the future diagnosis of the disease.

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* Med. Chir. Trans., vol. xix., 1835.

† Dublin Journal, vol. xiii., p. 142, 1841.

§ II.—DILATATION.

895. The most noteworthy example of this state, I know of, has been recorded by Townsend of Dublin. An emaciated man, aged sixty-one, who had for upwards of a year mainly suffered from paroxysmal dyspnœa (attended with percussion-dulness of the chest, and notably relieved by bleeding), free from rigors, night-perspirations or diarrhœa; with a pulse of 100, feeble and very soft, but regular; the cardiac movements and sounds excessively weak; the respiration null over the left lung, exaggerated superiorly, but very weak inferiorly, in the right organ; dies asphyxiated after five weeks' stay in hospital. The lungs, emphysematous anteriorly, were excessively heavy and fluctuated distinctly. On cutting into the left lung three pounds and a half of blood poured forth. The pulmonary veins furnishing this blood reached at least four times the normal calibre, from the smallest radicles to the large trunks,—the latter forming capacious sacs outside the auricle. The same condition, less developed, affected the veins of the right lung. The dilatation had arisen from pressure of tuberculous infiltration of the wall of the left auricle on the veins at their line of union with that cavity; so extreme was the pressure, that a sound could with difficulty be introduced through the veins into the auricle. The right cavities of the heart (otherwise healthy) were dilated. The bronchial glands had undergone extensive, the lungs very slight, tuberculisation.*

§ III.—RUPTURE.

896. A man, aged twenty-one, perishes in a fit of hæmoptysis, after three years' suffering from symptoms of diseased heart. The pericardium was found adherent; both sides of the heart greatly dilated; the aortic valves obstructively diseased to a grave amount. The immediate cause of death was traced to a rupture, nearly an inch long, of one of the left pulmonary veins, close to its point of issue from the lung; the left pleural sac was distended with blood.†

§ IV.—CANCER.

897. The trunks and medium-sized branches of the pulmonary

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* Dublin Journal, 1832.

† Portal, Mém. de l'Acad. des Sciences, Paris, 1784.

veins occasionally contain, in cases of cancerous disease of the parenchyma, more or less commingled fibrinous coagula and carcinomatous substance. This accumulation may occur in association with, or independently of, a similar state of the pulmonary artery.

Such obstruction of the interior of the vessel, if to any notable extent, must greatly increase the dyspnœa arising from infiltration of the parenchyma; but I am unacquainted with any means by which obstruction of the kind could be diagnosed,—and more especially by which it could be distinguished from that much more common condition, obliteration of the veins by pressure of cancerous substance on the outer surface.

§ V.—THROMBOSIS.

898. Edward Smith has placed on record the case of a lady, aged twenty, who, being in apparently excellent health, and within a few days of her expected confinement of a second child, while lying, dressed, on the bed, suddenly uttered a shriek, threw her arms about, crying wildly for air, then became calm for a moment, saying she was "better," and expired. At the moment of death the face was *deeply livid,* and the body bent. Forty hours after death: slightly sanious fluid had escaped from the nostrils; posterior vibices; no swelling of the legs; blood black and universally fluid, "except in the *pulmonary veins,* where the whole tube was filled by a cylinder of coagulum, not strongly adherent to the lining membrane of the vein." Heart flaccid, left cavities empty, right ventricle containing, right auricle distended with, black fluid blood; valves healthy. Arteries preternaturally small; no blood in any of them. Veins universally distended, their calibre about as much above, as that of the arteries below par. No venous or arterial rupture anywhere. Stomach and intestines enormously distended with gas. Lungs collapsed, but faintly crepitant. Sinuses and large cerebral veins full; nothing abnormal in the brain. The white corpuscles are said to have been in considerable excess,—but there is no evidence that the increase had exceeded that usually occurring in pregnancy. The absence of any mention of the *pulmonary artery* in the narrative is most singular,—and, taken in connection with the loaded state of the right and empty state of the left cavities, increases very seriously the difficulty of interpreting the mechanism by which life was immediately arrested. The narrator is inclined to look

upon the pulmonary venous coagula "as a cause and not merely an attendant occurrence of the death."* The case illustrates the truth that the phenomena of syncopal and asphyxial death are closely allied.

899. The pulmonary veins become the seat of murmur, continuous and remittent, in cases of intense spanæmia: this murmur is audible in the back on each side of the mid-dorsal spine,† and may simulate in front, about the basic region of the heart, intracardiac diastolic murmur [629]. But whether any such continuous hum could be traced in, and might lead to at least the surmise of, coagulation within the pulmonary veins, remains to be determined.

SECTION V.—DUCTUS ARTERIOSUS.

§ I.—PATENCY IN THE ADULT.

900. If Alvarenga be right as to the frequency with which the Arterial Duct remains open to a more or less advanced period beyond childhood [827], the inference seems unavoidable, that such patency must often fail to produce demonstrable signs or symptoms. The acute observation of the present day would, otherwise, often have revealed anomalous physical conditions in the supra-basic region, as well as attendant symptoms, irreconcilable with the notion of their depending on the ordinary forms of cardiac and arterial diseases. Whether this be the true explanation or not, the fact remains, that clinical histories of open arterial duct are few and far between.

(*a*) Barlow and Babington give the main particulars of a case, in which cyanosis is referred to a patent state of the arterial duct in a woman cut off at the age of thirty-four. A variety of other intracardiac and arterial morbid states existed; and, further, the case appears to have been one rather of direct communication of the pulmonary artery and aorta in the site of the duct, that an example of openness of that canal. The effects would, however, be probably identical in the two cases. Not the least remarkable part of the matter is, that T. W. King, "learning the patient was a seven months' child, *then* diagnosticated patescence of the ductus arteriosus." It is, unfortunately, not stated on what specific grounds the diagnosis was made.[*]

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* Case read before Med. Soc. of London.

† Davis, U. C. H., Females, vol. iv., p. 138, 1848.

(*b*) A female, aged twenty-seven, presented the ordinary physical signs of dilated hypertrophy of the right compartments of the heart, with distended cervical veins and cyanotic discoloration of the skin and visible mucous surfaces. No murmur was to be distinguished over either ventricle, or at the mouth of the aorta; but within the pulmonary artery area (and especially towards the insertion of the third left rib), along with a strong second sound, was heard a double murmur, prolonged and filing in quality. The patient seems to have perished of general dropsy and bronchial catarrh, shortly after these facts had been ascertained. Examination disclosed, in addition to the expected hypertrophy, an open state of the arterial duct and of the foramen ovale, with dilatation of the pulmonary trunks. The open state of the foramen and duct does not appear to have been suspected during life.[†]

(*c*) Hilton Fagge records the case of a female, aged forty-two, in whom a diastolic "wavy" murmur, loudest about the inner part of the pulmonary cartilage (where its quality was musical), scarcely audible elsewhere, and certainly not carried down the sternum, followed the second, while separated by a considerable interval from the first, sound. There was besides a systolic blowing murmur, strongest in the fourth interspace close to the sternum. The pulse ranged from 34 to 43. The supra-basic diastolic murmur was hypothetically referred by the observer either to flow of blood through a communication between the pulmonary artery and aorta, or to the systole of an auricular appendix rendered hypertrophous through mitral stenosis.

The patient came again under notice, three years later, with the same infrequent pulse, and a loud systolic murmur at the apex: but the peculiar diastolic *supra-basic* [‡] murmur, "if audible at all, was exceedingly faint." A few days later, death occurring (in the main from disconnected ailments), cardiac hypertrophy, chiefly on the right side, great dilatation of the pulmonary artery, closed foramen ovale, with a ductus arteriosus, forming a short canal sufficiently open to admit a No. 6 catheter, were discovered.

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[*] Patholog. Trans., vol. i. p. 55, April, 1847.

[†] Prager Vierteljahrscrift, vol. lxxiii., p. 92, 1862. Case reported by Kaulich from the clinical practice of Jaksch.

[‡] As reported in Guy's Hospital Reports, vol. xviii., 1873, it would appear as if the special murmur had only been sought for on the second occasion "at the *apex;*" but obviously the author must have written *apex* instead of base by a *lapsus calami*, as he had no reason, on his own showing, to expect to find the special murmur in the former position.

901. *Diagnosis.*—From these cases it would seem to follow, as a matter at the least of fair conjecture, that, if a cyanotic adult presented, first, the signs of decided right cardiac hypertrophy, secondly, a negation of murmur at either apex of the heart, thirdly, a single prolonged diastolic or a double murmur, of maximum force at the pulmonary cartilage, and not conducted downwards, the cause of those combined conditions would be found in a patent state of the ductus arteriosus.

Why had the murmur quasi-disappeared in Hilton Fagge's case at the second time of observation? Unless supposed to be the effect of *præ-mortem* feebleness, the occurrence is unsatisfactory in its diagnostic signification.

§ II. ANEURISM.

902. The earliest recorded example of aneurismal duct appears to be that of Billard: the essential facts were as follows:—

A male infant died on the third day of its existence with lividity of face, difficult respiration, stifled cry, small, weak, frequent pulse, but natural temperature of the surface. The ductus arteriosus, dilated to the size, and in the form, of a cherry-stone, was almost filed with clots,—a passage, sufficient to admit a crow-quill, remaining pervious. The rest of the body presented nothing remarkable.*

903. The diligent search of Thore, some while since, led to the discovery of two other cases, and his own note-books supply eight others, furnished by upwards of 1000 *post-mortem* examinations made by himself at the Enfans Trouvés of Paris.† From these data some few general inferences may be drawn.

904. The form of aneurism seems to have been always either of the fusiform or globular species of dilatation (Diag. p. 476, figs. 1 and 2); the coats of the duct remaining unbroken. The dilatation seems almost always to occur nearer to the aorta than the pulmonary artery. In ten of the eleven cases, clots, either lumpy or stratified more or less completely, filled the vessel; in one the contents were liquid. In no instance does the dilated duct appear to have exceeded a very small nut in size.

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* Billard, Mal. des Enfans Nouveaux-nés, Paris, 1828. The case was observed in 1826.

† Archives Gén. de Médecine, 1850, p. 30.

In no one of the series of cases was any malformation of the heart discovered.

905. In eight of the eleven cases the patients were males; in two, females; in one instance the sex is not mentioned.

906. Death occurred at various times, between the first twenty-four hours and the nineteenth day.

907. Thore maintains that, even if the blood-current continue to pass through the duct, and *à fortiori* if it be closed by thrombi, no symptoms indicating disturbed circulation would, "at all events for some time," ensue. And, as far as the evidence of these cases reviewed goes, death was in no wise hastened by the morbid condition of the duct.

APPENDIX.

Vide p. 13.

WEIGHT OF THE HEART.

According to Boyd's measurements the weight of the heart, from birth to the close of life, stands at various periods, and in the two sexes, as follows (Aitken's Science of Medicine, vol. ii., p. 701, 2nd Ed.) :—

Ætatis.		Ounces.	Ætatis.		Ounces.
1 to 2	M.	1.66	30 to 40	M.	11.36
	F.	1.47		F.	9.45
2 to 4	M.	2.14	40 to 50	M.	11.53
	F.	2.11		F.	9.60
4 to 7	M.	2.77	50 to 60	M.	11.83
	F.	2.30		F.	10.44
7 to 14	M.	4.25	60 to 70	M.	12.94
	F.	4.38		F.	10.64
14 to 20	M.	7.61	70 to 80	M.	13 14
	F.	8.46		F.	10.10
20 to 30	M.	10.06	Above 80	M.	12.10
	F.	9.08		F.	10.27

Vide pp. 173, 180.

EMETICS IN PALPITATION.

The utility of emetics in the treatment of palpitation, when the stomach is loaded with food or with acid secretions, is matter of common knowledge. But emetics will occasionally prove the sole agents capable of arresting a fit of the most irregular palpitation, when there is no notable accumulation, solid, liquid, or even gaseous, in the stomach; the eructation of a little flatus, nay, even the mere act of retching, will sometimes put an instantaneous stop to the heart's disturbance. Is there some specific influence exercised on the vagus nerve by the emetic in such cases, which restores its temporarily paralysed inhibiting force? Whatever be the explanation, however, the clinical *fact* remains.

Vide p. 242.

ALCOHOL IN ACUTE INFLAMMATION.

The warning, against the abuse of alcohol in the treatment of acute inflammations, which is given in the page referred to, receives ample justification in a recent essay by J. P. Heslop ("The Abuse of Alcohol in the Treatment of Acute Diseases," 1872). In an able analysis, statistical and argumentative, of the cases of the late R. B. Todd, this writer most satisfactorily disposes of the fallacy that the King's College Professor had struck out a plan for the management of pyrexial inflammation either superior to, or even as safe as, various others less heroic than itself.

Vide p. 512.

IODIDE OF POTASSIUM IN ANEURISM.

Although it be perfectly true, as stated in the text, that there seems to be what may be termed an *aneurismal power of toleration* of the drug, still I have known the testes waste under its free use in aneurismal sufferers; under the circumstances, the *loss* may, perhaps, be considered a *gain*.

RADIAL SPHYGMOGRAMS.

For the series of pulse-tracings, contained in the plates, I am indebted to Mr. F. A. Mahomed, whose well-known manipulative skill in all that concerns the sphygmograph, is a guarantee of their accuracy. In order to insure the fidelity of their reproduction, the original tracings have been photo-lithographed.

PLATE I.

Fig. 1 (*vide* pp. 126—129). *Healthy pulse* of a moderately strong man; pressure employed = 1 oz. only. Percussion, tidal and dicrotic waves all just discernible; no tricrotism. Upstrokes and downstrokes equal; apex-line and base-line horizontal.

Fig. 2. Variety of pulse of people *free from cardiac disease*, but leading sedentary and luxurious lives; occurs in excessive smokers. Percussion-wave imperceptible; dicrotic wave [208] extremely marked. Pressure = 2 oz.

PLATE I.

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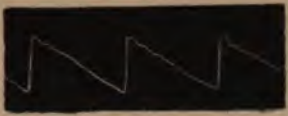

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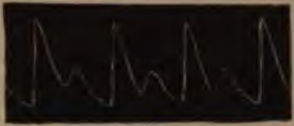

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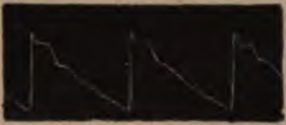

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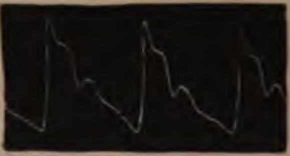

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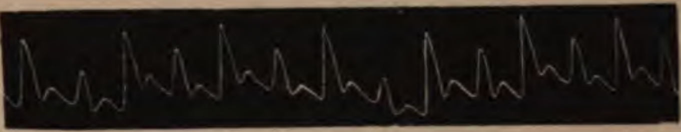

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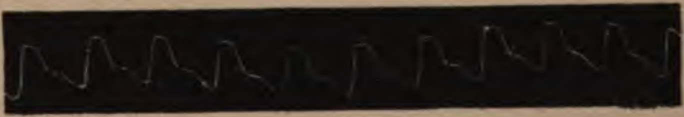

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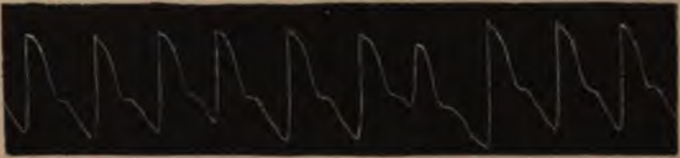

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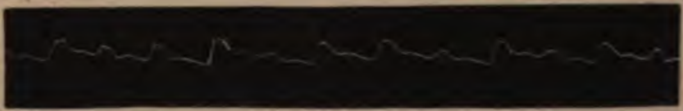

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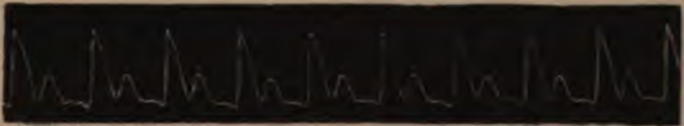

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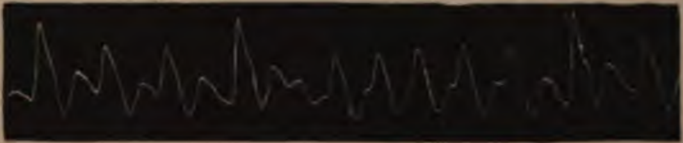

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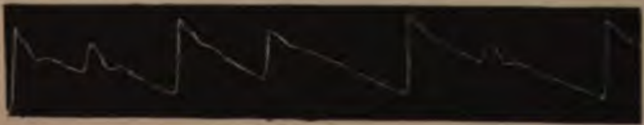

[illegible] Holborn, London.

Fig. 3. *Regular pulse* of moderately strong man, *ætatis* 56, free from organic disease of any kind. Pressure = 1½ oz. Notched and square-topped appearance, caused by sustained tidal wave, in turn the effect of failing elasticity of the vessel-walls. Faint tricrotism is perceptible. The tracing exhibits, in a minor degree, the characters of the pulse of calcification [773],—which, as Lorain has distinctly shown (Op. cit. p. 249, Fig. 328), closely resembles that of some examples of aortic reflux.

Fig. 4 (*vide* p. 287). *Pulse of ventricular hypertrophy with some dilatation of the cavity*: pressure = 3 oz. Characteristics: height of upstroke; hooked apex (there is no diastolic basic murmur,—therefore explicable as in Fig. 3); sustained tidal wave, result of prolonged ventricular contraction and full volume of blood propelled; dicrotism well, tricrotism very slightly, marked. Pulse regular in rhythm and force.

Fig. 5 (*vide* p. 366). Variety of pulse of mitral reflux, with dilated hypertrophy of both sides of the heart, in a male aged forty-two. Soft, short, inconstant, systolic murmur at left apex and axilla. Heart found to weigh 19 oz. Mitral orifice nearly admitted whole hand, valves, not having increased *pari-passu*, were totally *insufficient*. Variable amount of distension of the orifice, with different systoles, may have rendered the valves variably incompetent, and so allowed, or not allowed, of the production of murmur. Characters of the tracing (pressure = 4 oz.): suddenly collapsing tidal wave, indicating small quantity of blood sent onwards by each systole; great amount of dicrotism [208]; regularity of rhythm [607]; irregularity of force and volume; extreme unevenness of the respiration-line [211], resulting from distension of the heart and pulmonary obstruction.

Fig. 6 (*vide* p. 278). Pulse of woman, ætatis 40, suffering from *progressive muscular atrophy*. Characters (under pressure = 6 oz.): absence of percussion; slight obliquity of upstroke; flattened apex and prolonged tidal wave; unevenness of respiration-line. Obviously the heart was (from the last character) oppressed; the ventricle contracted slowly,—though, as appears from the pressure employed, not feebly. There was no murmur; the apex-beat not defined; impulse perceptible outside nipple; cardiac sounds full-toned as of dilatation. The real condition of the fibres of this heart may be questioned. Was there fatty change of hypertrophous and dilated ventricle?

Fig. 7 (*vide* p. 287). Pulse of hypertrophous left ventricle and inelastic arteries in a hard-drinker (variously diseased), ætatis 60; deep cardiac dulness in excess; sounds wanting in tone; no murmur. Characters (under pressure = 4 oz.): high upstroke; tidal wave, reaching apex, too prolonged; moderate dicrotism; occasional (dynamic) irregularity of force and rhythm.

Fig. 8 (*vide* p. 365). The small, irregular, unevenly feeble variety of pulse of mitral reflux [607, *c*], with dilatation and thickening of the ventricle, and a functional condition tending to *asystolism*. Left apex systolic murmur, audible in vertebral groove (p. 94). Rheumatic male, aged 52. Characters (under pressure = 3 oz.): Upstrokes short, oblique, and of unequal length; percussion absent and tidal wave small; amount of dicrotism, always slight, varies in different pulse-curves; base or respiration-line [211] very slightly uneven,—a condition observed, where the disturbance of the left heart and systemic circulation becomes so extreme that the respiration-act fails to affect arterial tension.

Fig. 9 (*vide* p. 365). Another variety of mitral reflux pulse with dilated hypertrophy,—systolic murmur at left apex and in vertebral groove. Mitral cusps ulcerated, shortened, inefficient, with fibrinous attached shreds (the murmur had been occasionally musical (p. 83)). Characters (under pressure of 5 oz.): percussion-wave fairly marked; tidal wave of good size, though short and unsustained; dicrotism highly marked, from systemic, venous, and capillary stasis; very slight tricrotism.

Fig. 10. Pulse of same case as Fig. 9. Heart in a state of ataxy and tending to asystolism, systemic obstruction greatly increased. Irregularity of force and rhythm extreme; hyper-dicrotism; increased tricrotism; unevenness of respiration-line.

Fig. 11 (*vide* p. 370). The statements made in the text concerning the tactile characters of mitral stenotic pulse are supported by the sphygmograms printed by Lorain (Op. cit. p. 290), especially as regards the fact of habitual regularity being converted into irregularity by excitement and respiratory disturbance. Distinctive characters are in truth wanting, unless the peculiarity in Fig. 11, as originally maintained by F. A. Mahomed (Med. Times, May 18, 1872) should eventually be proved to signify constriction of the mitral orifice. Characters (under pressure = 1 oz.): peculiar irregularity, caused by a "secondary" contraction, during ventricular diastolic time at regular intervals (in this case at every third beat),—the heart's rate of movement slackened by digitalis. That this contraction is really a "secondary" one, and not an ordinary small, irregular, inefficient contraction, seems shown by the facts, that it never commences from the base line but from a spot in the down-stroke, and that it is not followed by several small and inefficient contractions.

PLATE II.

Fig. 1 (*vide* p. 372). Tracing in a female, aged 55, with the ordinary signs of *tricuspid reflux*, dilated and pulsatile external jugulars, &c.

PLATE II.

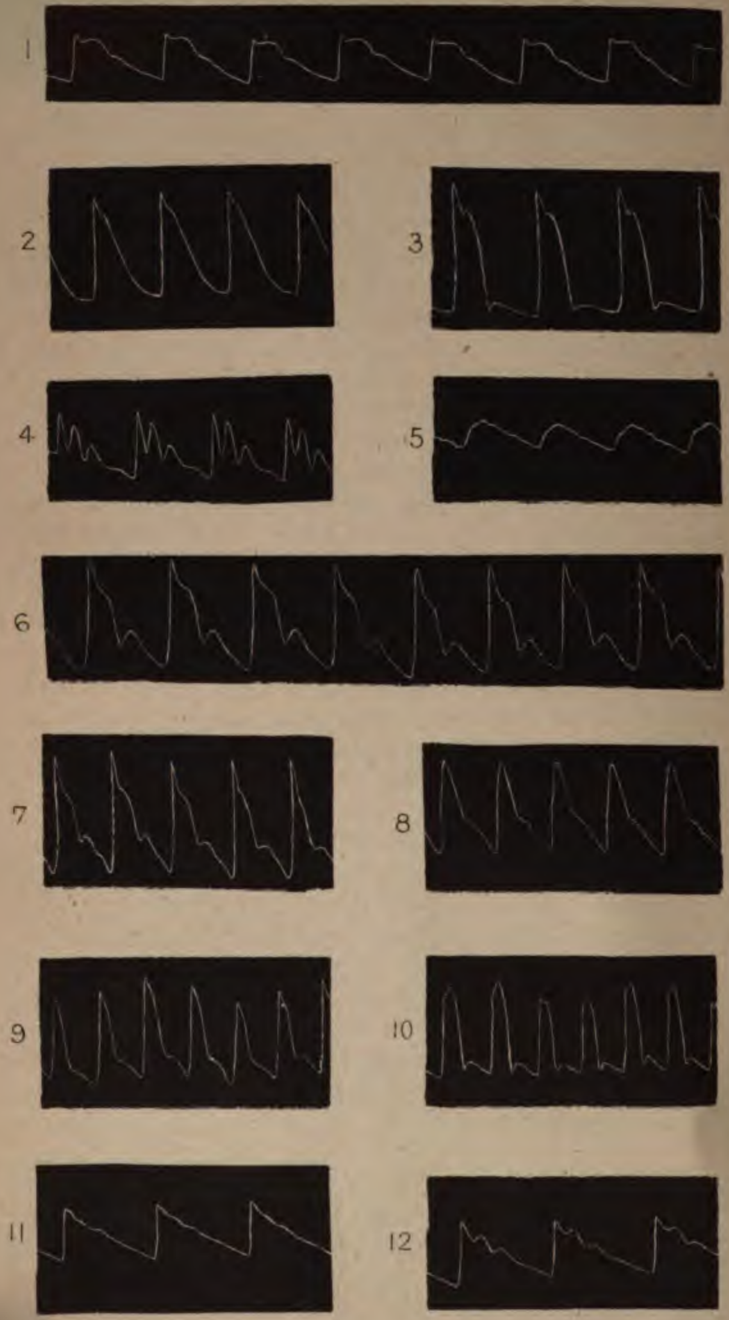

Photo-Lith. Whiteman & Bass 236 Holborn London.

Characters (under pressure = 2½ oz.): notched apex, sustained tidal wave with flattened top; very faint dicrotism, and questionable tricrotism. All this is without diagnostic significance. The base- or respiration-line often uneven, is here the reverse.

Fig. 2 (*vide* p. 381). Pulse of *free aortic reflux*, one cusp of the valve being entirely destroyed; left ventricle dilated and hypertrophous; aorta healthy: male, aged 34. Characters (under pressure of 3 oz.): well-marked percussion-wave, giving a sharp and long, hooked apex (less marked here, than often, on account of accompanying dilatation); large, full tidal wave; dicrotism totally absent. The hooking of the apex is, on the whole, slight, and varies in amount in different curves.

Fig. 3 (*vide* p. 381). Pulse of *aortic reflux with atheromatous aorta* in a female, aged 40. Characters (under pressure of 1 oz.): high vertical up-stroke; percussion-element well-marked; tidal wave square and topped with very sudden collapse; emptiness in diastole striking, still dicrotism distinct on small scale,—probably from venous systemic stasis [209]. As a rule, the typical characters are much better marked, if the arm be held vertically, than placed horizontally.

Fig. 4 (*vide* p. 377). Pulse of *aortic stenosis* extreme in amount but recent and unattended with hypertrophy; two cusps adherent to each other and fresh warty growths blocking up the passage. The essential character ascribed by Marey to the sphygmogram of aortic stenosis, and figured by Lorain (Op. cit. p. 272), namely, oblique upstroke, is wholly wanting here; the upstroke is sharply vertical,—and yet there was no hypertrophy. The polycrotous condition of the down-stroke seems inexplicable.

Fig. 5 (*vide* p. 377). Pulse of *aortic stenosis* of old standing: systolic basic, without diastolic, murmur. Characters (under pressure = 4 oz.): oblique up-stroke; percussion deficient; dicrotism faint or null.

Fig. 6 (*vide* pp. 287 and 443). Pulse in a pregnant woman, affected with a mild form of *cardio-thyroid exophthalmos*. Characters (under pressure = 4 oz.): high vertical up-stroke; somewhat hooked apex (no diastolic basic murmur); rather sustained tidal wave; dicrotous wave well marked; curves identical; respiration-line even. These are the characters of associated hypertrophy,—and the sphygmogram may be compared with that in Fig. 4, Plate I.

Figs. 7 and 8 (*vide* p. 504). Radial pulses in a pure case of *aneurism of the innominate artery*,—the sac, about the size of an orange, gave origin to the subclavian and carotid arteries, while, on the proximal side, half an inch of innominate artery remained of normal size. Fig. 7. *Left radial.* Characters (under pressure = 4 oz.): those of hypertrophous ventricle with inelastic and calcified vessels, wholly unaffected by the aneurism. Fig. 8. *Right radial.* Characters (under pressure = 7 oz.) : absence of percussion and of dicrotism,—those waves being absorbed, as it were, by the sac.

In ordinary cases of innominate aneurism, the aorta being more or less involved, very slight, if any, difference is discoverable in the radial sphygmograms (p. 495).

Figs. 9 and 10 (*vide* p. 495). Radial pulses of *double aneurism of the arch,*—one sac in its ascending portion, the other seated opposite the origin of the left subclavian. A third aneurism (without influence on the radial arteries) was found in the descending portion of the arch. Fig. 9. *Right radial.* Characters (under pressure = 1 oz.) : up-stroke faintly oblique ; percussion-wave slight in comparison with the height of the up-stroke ; dicrotic wave feebly marked. Fig. 10. *Left radial.* Characters (under pressure = 1 oz.) : apex somewhat squared ; line of collapse of tidal wave almost perpendicular ; dicrotism, considerably more marked than in the right radial, *possibly* due to contraction of sac opposite the root of the left subclavian.

Figs. 11 and 12 (*vide* p. 522). Radial pulses in a case of *aneurism of the abdominal aorta :* the absence of disease of the thoracic aorta proved *post-mortem.* Fig. 11. *Right radial.* Characters (under pressure = 4 oz.): all attributes normal. Fig. 12. *Left radial.* Characters (under pressure = 4 oz.) : all the waves much more developed than in the left-side sphygmogram. The difference in the two sides was constantly present ; and seems to elude explanation.

Vide p. 485.

ACCENTUATED SECOND SOUND IN AORTIC ANEURISM.

The accentuation of the second sound, referred to in the text, is sometimes greatly more marked in one, or in both primary carotids, than in the arch of the aorta itself.

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